THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—22ND YEAR. SYDNEY, SATURDAY, MARCH 16, 1935. No. 11.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.
"Human Tuberculosis of Bovine Origin in Victoria", by REGINALD WEBSTER, M.D., D.Sc. .. 323
"The Pollen Content of the Melbourne Air During the Hay Fever Season of August, 1933-March, 1934", by MARJORIE M. SHARWOOD, B.Sc. 326
"Some Observations on the Ætiology of General Paralysis of the Insane", by C. R. D. BROTHERS, M.D., B.S. 332
"The Problems of Antenatal Care", by T. DIXON HUGHES, M.B., Ch.M., F.R.A.C.S. 334
"Thrombophlebitis Migrans, with an Illustrative Case", by MOSTYN L. POWELL, M.B., M.R.C.P. 336

REPORTS OF CASES—
"A Retrocaecal Appendix", by J. L. MEAGHER, M.B., B.S. 339

REVIEWS—
Experimental Physiology 339
The Hormones 340
Progress in Endocrinology 340

NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—
Materia Medica for Nurses 340

LEADING ARTICLES—
The League of Red Cross Societies 341

CURRENT COMMENT—
Cardiac Œdema 342
Cancer of the Bronchus and Lung 343

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—
Pædiatrics 344
Orthopædic Surgery 345

BRITISH MEDICAL ASSOCIATION NEWS— PAGE.
Scientific 346
Nominations and Elections 349

CORRESPONDENCE—
The Training of Surgeons 349
Treatment of Septic States Associated with Pregnancy 350
The White Man in the Tropics 350

UNIVERSITY INTELLIGENCE—
The University of Sydney 351

CONGRESSES—
Ligue Internationale Contre le Rhumatisme 351

PROCEEDINGS OF THE AUSTRALIAN MEDICAL BOARDS—
Queensland 351
Tasmania 351

MEMORIAL TO THE LATE NEVILLE HOWSE .. 351

BOOKS RECEIVED 351

DIARY FOR THE MONTH 352

MEDICAL APPOINTMENTS 352

MEDICAL APPOINTMENTS VACANT, ETC. 352

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 352

EDITORIAL NOTICES 352

HUMAN TUBERCULOSIS OF BOVINE ORIGIN IN VICTORIA.[1]

By REGINALD WEBSTER, M.D., D.Sc.,
Pathologist to the Children's Hospital, Melbourne.

IT is apparent that the field of human pathology most likely to yield information relative to the part played by the bovine tubercle bacillus in human morbidity is that dealing with diseases of children. This conclusion is obvious indeed and a necessary corollary to the dominating influence of milk in the diet of young children. In the course of my experience as pathologist to the Children's Hospital, Melbourne, I have made some studies on this question, the results of which were embodied in a paper published in THE MEDICAL JOURNAL OF AUSTRALIA of March 5, 1932.[1]

[1] Read at a meeting of the Australian and New Zealand Association for the Advancement of Science, Melbourne, January, 1935.

I know of only one other bacteriological investigation directed towards the elucidation of the rôle of the bovine tubercle bacillus as a factor in tuberculosis of childhood in this State, and that was a research by Dr. W. J. Penfold, the results of which were communicated to the Australasian Medical Congress in 1923 and published in the transactions.[2] With this work I was associated to the extent of collecting all the material on which Dr. Penfold's observations were based, and the net result is that all the recorded bacteriological work in this State concerning the bovine tubercle bacillus as an agent in the production of human disease is founded on material derived from the Melbourne Children's Hospital. This places the children examined all in one social group and defines fairly well their hygienic and economic status.

W. J. Penfold investigated 72 cultures from 60 patients,[2] and my own work embraced the study of 86 cultures obtained from 81 patients.[1] Our knowledge, therefore, regarding the extent of bovine

tuberculosis among children in Victoria is limited to the facts elucidated with respect to 141 children and the inferences permissible therefrom. Although the total of 141 is not very large, those familiar with the lengthy bacteriological technique required for the investigation of each specimen will allow that it represents a great deal of work; and I would further submit that it furnishes a reasonably reliable index of the share of the bovine tubercle bacillus in the tuberculosis of childhood. This claim is based on the general agreement in the findings of Penfold and myself, working quite independently, with an interval of nine years between the studies. Thus in the 60 cultures of *Bacillus tuberculosis* in Penfold's group there were 12 bovine and 48 human strains, an incidence of bovine infection of 20%. In my own series of 81 children, none older than fourteen years, and exhibiting varied clinical manifestations of tuberculosis, the incidence of bovine infection was 21, or 25·9%.

The number of bovine strains encountered is governed by two main factors: (i) the age of the child, and (ii) the nature of the pathological material examined. As illustrating the operation of the second factor, I would adduce the finding that in 24 samples of caseous detritus from tuberculous glands of the neck which I examined, the bovine bacillus was implicated in no less than 17, the remaining seven yielding *Bacillus tuberculosis* of human type. Tuberculosis of the tonsils of seven children was due to bovine infection in four instances and human in the other three. In a small group of mesenteric glands from four children the bovine bacillus was implicated in every one.

The close anatomical and physiological relationships of the tonsils and cervical glands demand their consideration as one group, and if to the 31 specimens of such group the four samples of mesenteric glands be added, a group of 35 is obtained in which the incidence of bovine infection reached the startlingly high figure of 71·4%.

The remaining 46 cultures examined were derived from a diversity of tissues, including bones and joints, bronchial glands, lung tissue and cerebrospinal fluid; and in not one of these situations did the bovine bacillus appear. Throughout the investigation the specimens of pathological material were taken as they came and no selection was exercised, so that the inclusion of all forms of tuberculosis in childhood had the effect of diluting down the remarkably high proportion (71·4%) of bovine infections in the tonsil-cervical gland group to the 25·9% which I found to represent the activity of the bovine tubercle bacillus in the tuberculosis of childhood.

On a priori reasoning it would be anticipated that a high incidence of bovine infection would be found in the tonsils, cervical and mesenteric glands. Tubercle bacilli ingested with infected milk are detained in tonsillar crypts or in the recesses of adenoid vegetations, whence they pass to the related cervical glands. It is important to note also that very frequently tuberculous lesions are induced in the tonsils and adenoids, as evidenced by the characteristic tissue reaction to tuberculous infection commonly displayed in histological sections of the tonsils of children affected with tuberculous cervical adenitis. Such tonsillar tuberculous lesions are scarcely, if ever, recognizable by the clinician, but that they cannot be ignored in the treatment of tuberculous cervical adenitis is self-evident. This question of tuberculous tonsillitis, very frequently due to the bovine organism, I have discussed very fully elsewhere.[3]

Australian observations on the type of tubercle bacillus responsible for bone and joint tuberculosis appear to be limited to 31 investigated cases (Penfold 4, Webster 27) and all were shown to be human infections. In my own series 22 patients were children and five were adults, and of the 22 children 10 were under five years of age—the period of maximum exposure to bovine infection. In 11 of the 22 children there was a definite and unequivocal history of intimate contact with pulmonary tuberculosis, but in the remaining 11 there appeared to be no phthisis in the child's family, and the source of infection could not be traced.

A formerly widespread impression, which is now, I think, largely dissipated, was that bone and joint tuberculosis was commonly due to the bovine bacillus. The belief gained ground on the reasoning that "local" or "surgical" tuberculosis assumed its particular clinical form because of the relatively low virulence of the bovine tubercle bacillus for the human subject.

Donald Paterson, in his book, "Sick Children: Diagnosis and Treatment", published in 1930,[4] makes the statement in connexion with bone tuberculosis that:

> Undoubtedly the bovine infection is much more frequently responsible than the human type, and bone tuberculosis is actually more prevalent in districts where the milk supply is infected.

This statement, of course, is to be read as applicable to Great Britain, but the first sentence is at variance with the findings of A. S. Griffiths,[5] who published a comprehensive review of 593 cases of bone and joint tuberculosis in Great Britain in which the type of infecting bacillus had been determined. In this large series the bovine bacillus was implicated in 20·5% of the total, the human bacillus being responsible for the majority. Under the age of five years the proportion of bovine infections was 32·8%, from five to ten years it was 24·5%. In subsequent age groups the incidence of bovine infection fell rapidly and no patient over twenty-three years of age yielded the bovine bacillus.

It would be unsound, of course, to draw a general conclusion regarding the amount of bone and joint tuberculosis occasioned by the bovine bacillus in Australia from the relatively small number of cases in which the necessary bacteriological examinations have been made, but from experience to date the suggestion arises that tuberculosis of bones and

joints among children in this country is to be traced much more frequently to the human vector than to the bovine.

Another group of tuberculous specimens examined consisted of bronchial glands and lung tissue obtained from *post mortem* sources. Of 27 cultures of *Bacillus tuberculosis* recovered from bronchial glands and pulmonary tissue, examined culturally and by virulence to rabbits, all were of the human type—classical eugonic strains of low virulence to rabbits. All the children from whom these *post mortem* specimens were obtained died of disseminated tuberculosis, and the autopsy findings were indicative of a respiratory mode of infection. The majority were very young children, 16 of 27 from whom cultures were obtained being under two years of age.

It was anticipated that human strains would predominate in this group, respiratory tuberculosis of bovine origin being very uncommon. W. T. Munro[6] reported the recovery of bovine bacilli from the sputa of ten patients in the course of seven years at the Fife and Kinross (Glenlomond) Sanatorium, and remarked that the recovery of the bovine bacillus from the sputum was sufficiently rare to command attention. While this is so, it would not have been surprising to have met bovine bacilli in the bronchial glands, as it is a generally accepted view that these glands may become involved secondarily from the mesenteric glands. Such was the experience of Penfold, who, in the paper[2] to which reference has been made, recorded two bovine and nine human strains recovered from *post mortem* specimens of bronchial glands. The same observer, reporting on 70 strains of the tubercle bacillus, isolated from adults and in almost every instance from the sputum, found all to be human in character.

In 71 samples of sputum examined in the Health Laboratories of the Commonwealth Department of Health[7] in the several States during the period 1924-1928, the human tubercle bacillus was identified in every instance.

It may be accepted, therefore, from the results of laboratory investigation that bovine tuberculosis is to be sought and the progress of the community in its elimination assessed in extra-respiratory infections, and particularly those of the tonsils and cervical glands. Holmes and Robertson, in the "Report on Bovine Tuberculosis in Man and Animals in Australia",[7] show clearly: (i) that the mortality from tuberculosis as a whole has shown a steady decline in Australia, and (ii) that the decline in mortality from non-respiratory tuberculosis has been much more pronounced than that in the respiratory group. Public health measures directed towards the control of the human vector should operate with equal incidence on both groups, and the inference would seem to be that the greater progress achieved in non-respiratory tuberculosis is the fruit of measures instituted for the control of bovine infection.

Such a conclusion is borne out by clinical experience. It is the unanimous opinion of all the clinicians at the Melbourne Children's Hospital, whose length of experience makes them competent to judge, that there is much less tuberculous cervical adenitis and abdominal tuberculosis at the present day than there was fifteen or twenty years ago. Clinicians claim, and with some reason, that the present-day operation of tonsillectomy, which has entirely superseded the older technique of tonsillotomy, has been one factor in the reduction of the amount of tuberculous cervical adenitis among the children of the community, but improved technique in the enucleation of tonsils would not go far if supervision of the milk supply were neglected.

The progress achieved in the reduction of non-respiratory tuberculosis, which, in the absence of universal routine bacteriological determination of the type of the infecting tubercle bacillus, furnishes our only index to bovine tuberculosis, is shown in the figures furnished by Holmes and Robertson.[7]

In the State of Victoria, for the period 1881-1890, the mortality rate per 100,000 of mean population from non-respiratory forms of tuberculosis was 35·3. For the year 1926-1927 this showed a decline to 9·2—a death rate of roughly one-fourth of that which obtained in the eighties. The figures for Queensland show still better results. Comparing the same periods, Holmes and Robertson found a reduction in the mortality rate from non-respiratory forms of tuberculosis from 53·0 per 100,000 of mean population to 2·4.

At first sight these figures seem very gratifying, but they do not by any means cover the whole question. There is indeed a danger that they will engender a feeling of unwarranted complacency, as they take cognizance of mortality only and leave out of account the question of invalidity. The fact that extra-respiratory tuberculosis, and especially bovine tuberculosis, has a relatively low mortality does not absolve us from the obligation to endeavour to abolish the enormous amount of invalidity for which it is directly responsible. Turning to laboratory results again, Penfold's figures[7] show that with respect to tuberculosis other than that of the respiratory system, 22% of the lesions in children yielded the bovine organism. My own results showed the incidence of bovine infection among all forms of tuberculosis exhibited by 81 children up to the age of fourteen years as 25·9%.

I would submit that it is not an exaggerated statement to make that if we could abolish bovine tuberculous infection, one-fourth of all the tuberculosis of childhood would disappear, with all its concomitant invalidity. It has been computed by Holmes and Robertson[7] that some 6,500 children between one and fifteen years of age have developed or will develop an infection of bovine origin, and if the rate of infection were the same in the other States as in Victoria, the resulting infections in Australia would total 25,000.

XUM

The veterinary measures to be taken towards the elimination of bovine tuberculosis may be left to those more competent to deal with them. When these are perfected, the problem for the medical profession, namely, to protect young children against infection from bovine sources, will cease to exist. In the meantime medical men have a choice of three lines of defence against bovine tuberculosis for the infants and young children in the families over which they exercise supervision.

In the first place they might rely on milk from tuberculin-tested herds and the guarantee that all reactors have been removed from such herds. This, in my opinion, is insufficient protection. There are many loopholes in the tuberculin test, and the largest perhaps is the varying potency and specificity of different commercial preparations of tuberculin. What faith I had in the tuberculin testing of herds was dissipated by an experience in which I obtained an indubitably bovine strain of tubercle bacillus from the cervical glands of a doctor's child who had never been given any milk other than a certain brand the name of which is synonymous with tuberculin-tested herds in this State.

Secondly, medical men might insist on the pasteurization of milk for infants and young children under their care. Theoretically, pasteurization should meet the situation. The thermal death point of the tubercle bacillus in milk has been investigated by various workers. H. Jenkins[8] has shown that milk containing large numbers of tubercle bacilli, artificially and naturally infected, is rendered non-pathogenic to guinea-pigs by exposure for thirty minutes to a temperature of 60° C., and that infected milk passed through a commercial pasteurizing plant is also disinfected in the same way. But it is doubtful whether pasteurization on a large scale for commercial purposes is uniformly effective, and bacteriological examinations of pasteurized milk sold as such have given reason to distrust commercial pasteurization.

The third course that may be taken is to urge parents and nurses to boil all the milk to be given to young children. It so happens that the most effective method of destroying tubercle bacilli in milk is also the simplest, but he who advocates routine boiling of milk raises storms of protest, chiefly concerning the scorbutic quality of boiled milk. Such defect is easily counteracted, and, were I a practising physician, I would urge the routine boiling of all milk in the dietary of infants and young children. The work reviewed in this paper affords the justification for the statement that one-fourth of all tuberculosis in childhood would be obviated by the universal adoption of this simple procedure.

References.

(1) Reginald Webster: "Tuberculosis in Childhood: The Incidence of Bovine Infection in Victoria", THE MEDICAL JOURNAL OF AUSTRALIA, March 5, 1932.

(2) W. J. Penfold: "Types of Tubercle Bacilli Prevailing in Australia", *Transactions of the Australasian Medical Congress (British Medical Association), First Session*, 1923, page 261 (Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, May 3, 1924).

(3) Reginald Webster: "Occult Tuberculosis of the Tonsil in Relation to Tuberculous Cervical Adenitis", THE MEDICAL JOURNAL OF AUSTRALIA, March 12, 1932.

(4) Donald Paterson: "Sick Children: Diagnosis and Treatment", 1930, page 436.

(5) A. Stanley Griffith: "The Types of Tubercle Bacilli in Human Bone and Joint Tuberculosis", *The Journal of Pathology and Bacteriology*, October, 1928, page 875.

(6) W. T. Munro: "Section of Medicine: Royal Society of Medicine", *The British Medical Journal*, May 4, 1929, page 813.

(7) M. J. Holmes and W. A. N. Robertson: "Report on Bovine Tuberculosis in Man and Animals in Australia", Commonwealth Department of Health Publication, 1930.

(8) H. Jenkins: "Experiments on the Pasteurisation of Milk, with Reference to the Efficiency of Commercial Pasteurisation", *Journal of Hygiene*, Volume XXV, 1926, page 273.

THE POLLEN CONTENT OF THE MELBOURNE AIR DURING THE HAY FEVER SEASON OF AUGUST, 1933–MARCH, 1934.[1]

By MARJORIE M. SHARWOOD, B.Sc.

(From the Baker Medical Research Institute, Alfred Hospital, Melbourne.)

DURING the warmer months in Australia pollen from various sources is plentiful, and it is becoming increasingly obvious that it is responsible for much discomfort and even some serious illness among those individuals who are specifically hypersensitive to it. The quantity of pollen present in the air on any particular day is apparently determined largely by the season and the weather. For instance, none has been found during steady rains, while it was plentiful in the spring, when the warm winds prevailed.

A great many articles have been published on pollen and its relation to hay fever and asthma. Blackley[1] in 1873 was the first to demonstrate that pollen was capable of producing the disease, and he was able to demonstrate a reaction to it on the skin and in the conjunctiva. His discovery was not put to any use until some years later. The other early workers were Dunbar[2] and his co-workers in Germany, and Noon[3] and Freeman[4] in England.

Several workers in America have made observations on the quantity of pollen in the air throughout the year. In his publications Scheppegrell[5] gives descriptions and microphotographs of the pollens commonly causing hay fever in America. He attempts to classify them into groups for immunological purposes and gives a list of the important pollens for several different districts. He also studied the density of pollen in the atmosphere at different heights. In 1916-1918 and 1922 he made surveys of ragweed pollen in New Orleans.

Since that time many surveys have been made in all parts of America.[6-22] Durham's work is the most extensive. He compares counts made for different parts of the United States of America, and also the ragweed incidence during several years.

[1] Read at a meeting of the Australian and New Zealand Association for the Advancement of Science, Melbourne, January, 1935.

Patterson and Gay compared the number of allergic patients having attacks with the number of pollen grains in the air and found that in the case of ragweed there was a close correspondence.

Maxwell[23] has enumerated the plants causing hay fever in Victoria, and work done in the asthma clinic of the Alfred Hospital confirms his results.

Derrick[24] in 1929 published a useful table of the time of pollination of various grasses in Victoria, but it was thought that some attempt should be made to estimate the actual amount of pollen available in the air. The work was facilitated by articles on identification of pollens by various workers, but especially by that of Moore and Garde in 1926.[25]

Method.

As a preliminary test, two microscopic slides coated with glycerine were exposed to the air for twenty-four hours—from 9 a.m. to 9 a.m. They were examined immediately afterwards. A cover slip seven-eighths of an inch square was placed over the centre and held in position with hard paraffin. The pollen grains were counted over this area. Types were distinguished on morphological grounds only, not by any chemical tests or staining. No attempt was made to distinguish between the pollen grains of various grasses.

The counts were begun on August 1, 1933, and were continued twice a week until the winter. For each day the larger of the two counts was always taken, this usually being that of the slide exposed to the prevailing wind. This method of collecting the samples was similar to those used in America. A different method is being used this year, which should give more accurate results. A wind vane, kindly lent by the Weather Bureau, was modified so that a microscope slide can rest at an angle of 45° under an aluminium shelter at one end of the horizontal bar. An opening behind the shelter enables the wind to blow through freely. The other end of the bar is fitted with an aluminium tail. By this means the slide is always exposed to the prevailing wind and is well protected from rain.

Pollen Morphology.

Control specimens of pollen of the commoner hay fever plants were mounted for identification purposes. The method described by Wodehouse[26] was adopted. A little of the pollen, from anthers previously unopened, was placed on a slide in a drop of methylated spirit. It was spread over the slide with a needle and then heated over a Bunsen burner for a few seconds to evaporate the methylated spirit. A drop of melted glycerine jelly or Canada balsam was placed in the centre with a cover slip. The glycerine jelly is really better for examination purposes, as the balsam is rather inclined to break the grains. It was found that the points of distinction are quite easily seen without staining.

Description of Pollen.

The following is a description of the pollen (principally after Moore and Garde):

(*a*) *Grasses—Gramineæ.* The distinguishing feature of the Gramineæ is the possession of one germ pore. They are often elliptical in shape, and the exterior surface is always smooth. The extine, or outer coat, is always narrow, except in the vicinity of the germ pore. The contents are never coarsely granular and are always colourless.

(*b*) *Plantain—Plantago lanceolata and Plantago major.* The pollen grains are always spherical, with smooth surface and of a pale yellow colour. Those of *Plantago lanceolata* have twelve germ pores, those of *Plantago major*, six. All the pores have lids, which are visible as small raised areas.

(*c*) *The Docks—Rumex* spp. The pollen grains of the docks are large, smooth-walled, and show four germ pores, without lids. The extine is narrow, but the contents are very coarsely granular. The germ pores usually give a square appearance to the whole grain.

(*d*) *Sorrel—Rumex acetosella.* The grain of sorrel is essentially similar to those of the docks, but only about half their size.

(*e*) *Composites—Compositæ.* The pollen grains of the Compositæ commonly found here are covered with long spines. These grains may be triangular, spherical or oval. Very few were found to be wind-borne.

Trees.

1. *Pine.* The grain of the English pine pollen is spherical, its distinguishing characteristic being two balloon-like wings.

2. *Cypress.* The pollen grain of cypresses is polyhedral, with a particularly thick outer coat, but with no wings.

3. *Oak.* The grain of the oak has three germ pores, the lids of which are extended into cones. These and the yellow colour are a distinguishing feature.

4. *Elm.* The grain of the elm is polyhedral, with a thick, even extine. The germ pores (five) are not easily seen, but the shape is characteristic.

5. *Eucalyptus.* The grain of eucalyptus has very definite triangular surfaces, with a very thick extine, and is quite colourless throughout.

6. *Acacia.* The whole grain of acacia is divided up into segments, giving the appearance of a bale of wool, and is quite characteristic.

Results.

Charts I, II and III illustrate the influence of weather upon the amount of pollen present in the atmosphere. The figures relating to rain, humidity and wind were obtained from the Weather Bureau.

The effect of rainfall is illustrated in Chart I. It can be seen quite clearly that heavy rain is associated with a low pollen count, as on October 5, November 30, December 15, January 9 and April 4. The only exception to this in fact is January 30, but the rain recorded on that day did not fall until the evening, and apparently the pollen was collected

on the slide before the rain fell. There were a few days when only a little rain was sufficient to cause the pollen count to fall, such as September 1, small one. One would expect the count to fall when the humidity is high, since the water would condense on the grains and precipitate them. This seems to

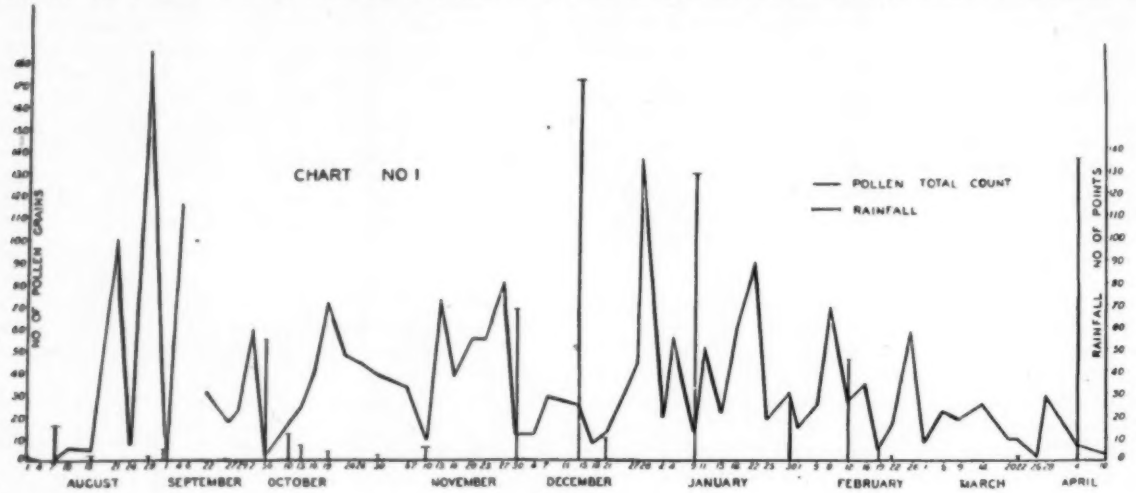

Chart I.

November 10, February 19 and March 26. But on other days a little rain appeared to have no effect at all, as on October 10, October 13 and December

be the case here to some extent, but it is not very definite, or if so the fall can be accounted for by the rainfall. But there are days when the count rose

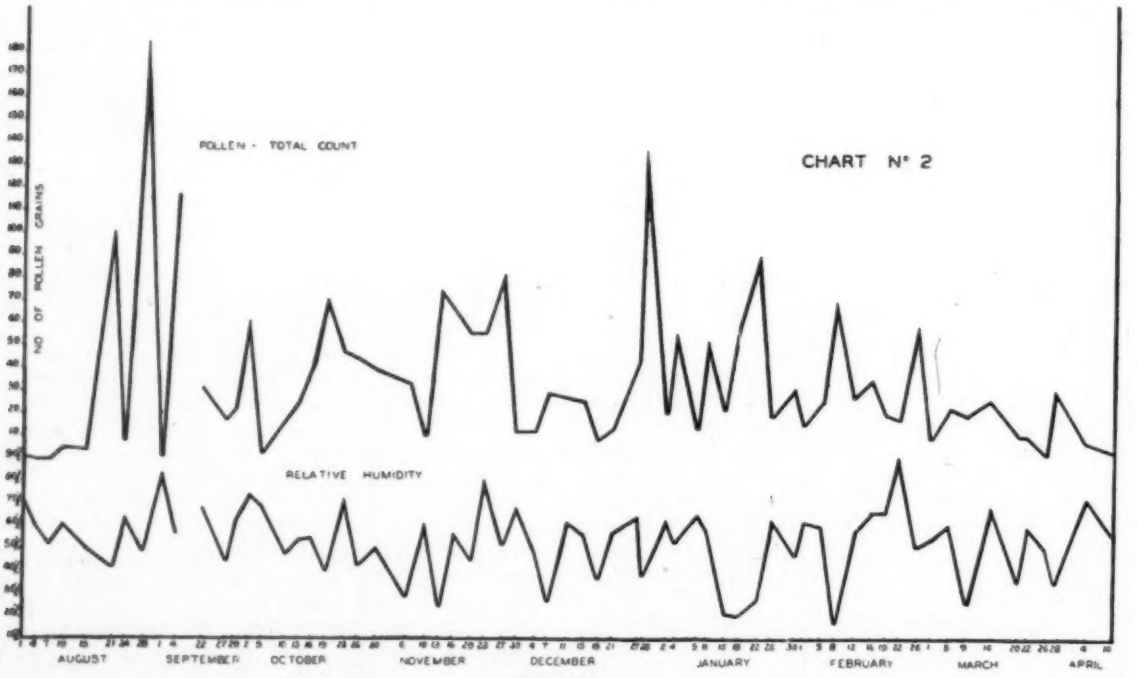

Chart II.

21. On these occasions also apparently some pollen was collected before the rain commenced.

The effect of the relative humidity is illustrated in Chart II, from which it appears that it is a very

with the humidity, such as October 2 and December 27. One would also expect the count to rise when the humidity is low, since a dry atmosphere favours the liberation of pollen. This is the case on

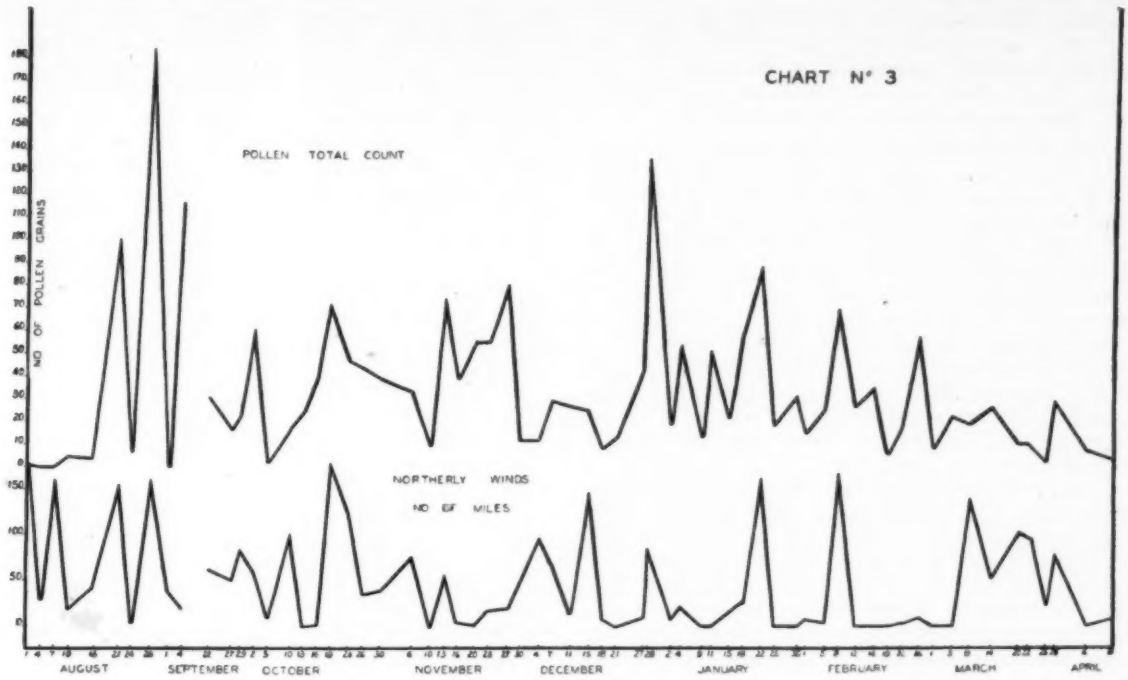

Chart III.

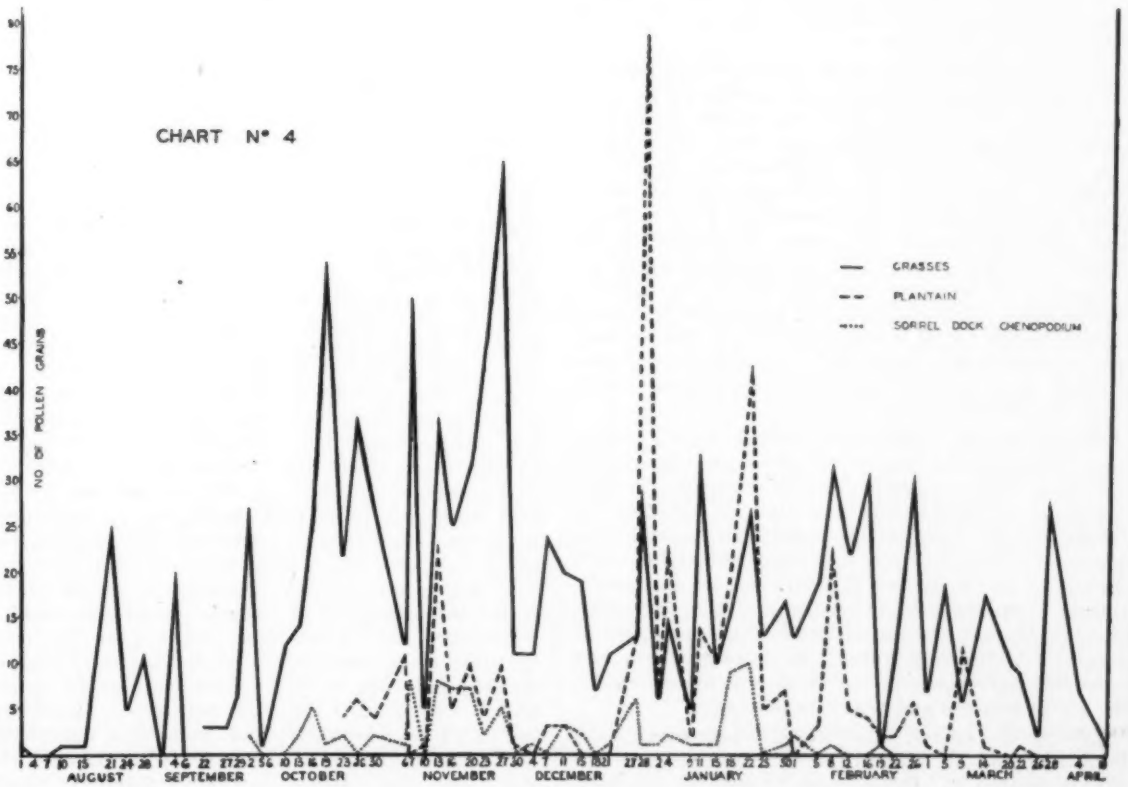

Chart IV.

December 28, January 18 and February 8, but there are three days when the count fell with the humidity, namely, September 27, December 18 and January 15. However, this chart is not complete, since observations were not made every day.

Chart III correlates the amount of pollen present in the atmosphere with the quantity of northerly winds operating. The speed and direction of the wind were observed every hour during the twenty-four. The number of times that northerly, north-easterly and north-westerly winds were found to be blowing at these times of observation was recorded. The speeds of the wind at these same times were added together, so that the first figure gives us an idea of the duration of the northerly winds and the second gives us an idea of the amount of it. The second figures were graphed and compared with the pollen count. Peaks in the graph are usually accompanied by a high pollen count. Exceptions to this occur on December 4 and 15, but this is because of the rain. In March also northerly winds were prevalent and pollen counts were low, but this can be accounted for by the dying off of the grasses and weeds. On the other hand, the pollen count rose quite considerably on several days when hardly any northerly winds were recorded. Such days are October 2 and November 27, which were fairly calm, and February 26, when a strong south-east wind was blowing. So it can be concluded that strong, dry northerly winds are very favourable for a large amount of pollen to be present in the air, but that they are not essential.

The amount in the air of the pollens of the grasses and of some weeds is illustrated in Chart IV. The grasses had the longest flowering period, beginning early in August and continuing until the winter. Their pollens gradually increased in number, reached a maximum in the latter part of November, and decreased again during December. This decrease was probably due to the rain, as illustrated in Chart I. There was a definite increase again in January and February, possibly due to the pollination of other varieties of grass, gradually declining during March. At the beginning of April the number fell rapidly and slides were no longer exposed. Possibly grass pollen would still have been found after that date.

The plantain pollination period began much later, towards the end of October, and its pollen was apparently present in the air all through November. There was a distinct decline in December, however, corresponding with that of the grasses. Its pollen was not really plentiful until the end of December, when it attained its maximum. During January and February and the first fortnight of March substantial but falling quantities of plantain pollen were found. In January there was a general agreement in the variations of content of the air of grasses and plantain pollens.

The weeds, sorrel, dock and chenopodium, have been included in one graph. The first two are closely related and their flowering periods are probably the same. Chenopodium has rather a later flowering period. The total number of grains found in the air was never very large. Sorrel and dock pollens began appearing on the slides at the beginning of October, and from then were found fairly constantly until the middle of January. The quantity was never very high at any time, but there was a maximum in November. Chenopodium pollen began to appear in November, but only very occasionally. It showed its maximum in January, and none was found after the middle of February.

Chart V shows the analysis of the tree pollens found in the air from August to November, 1933.

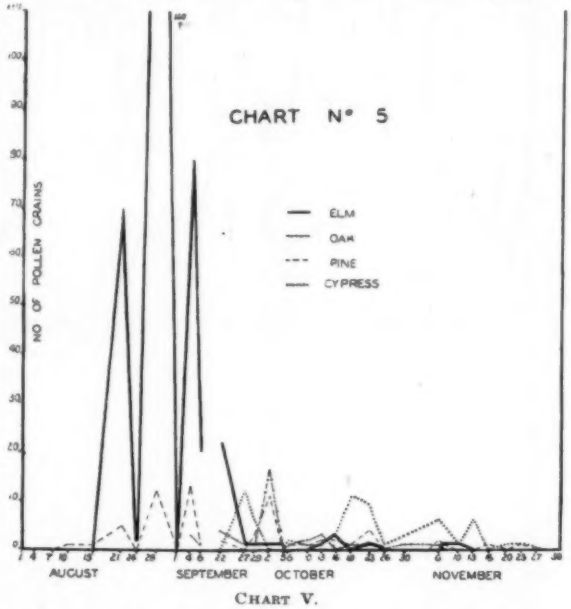

CHART V.

The large figures obtained in August are outstanding, but this can be accounted for by the fact that the hospital grounds and also adjoining parks are planted with elm trees. Elm pollen appeared in large quantities quite suddenly during the third week in August, and the same happened again on two other days, but after the third week of September only very little was obtained, and one or two grains were collected occasionally until the beginning of November. No oak pollen was collected on the slides until after September 22, and then only a very small amount, but one or two grains kept appearing until the beginning of November. Probably there were not sufficient oak trees in the locality to have much effect upon the pollen count. Pine pollen was appearing constantly on the slides from the beginning of August until the end of November, and increased in quantity on the days that elm was plentiful. After the beginning of October only one or two grains were found on the slides. Cypress pollen did not appear until the beginning of September, and from then was found fairly constantly until the end of November. The oak, pine and cypress are all present in small

Types and Quantities of Pollen Collected on the Days Specified.

Date.	Total Count.	Gramineæ.	Compositæ.	Plantain.	Sorrel.	Dock.	Chenopodium.	Pine.	Cypress.	Oak.	Elm.	Eucalyptus.	Acacia.	Doubtful.
1933—														
August 1 ..	1	1												
4 ..	0													
7 ..	0													
10 ..	5	1	2					1						1
15 ..	4	1						1						2
21 ..	100	25						5			70			
24 ..	7	5									2			
28 ..	184	11						12			160		1	
September 1 ..	0													
4 ..	116	20						13	3		80			
22 ..	31	3						2		4	22			
27 ..	17	3							12	1	1			
29 ..	22	6						3	6	1	1			5
October 2 ..	60	27			2			11		16	1	1	1	1
5 ..	2	1									1			
10 ..	17	12	1					1	1	2				
13 ..	25	14				2		2	1	3	1			2
16 ..	41	26	2			5		2	8		3			
19 ..	71	54				1		1	11	1				3
23 ..	47	22	4	4		2		4	9	1	1			
26 ..	44	37		6					1					
30 ..	39	27		4		2			3	1				2
November 6 ..	33	12		11		1		2	6	1				
10 ..	9	5	1	1					1		1			
13 ..	74	37		23	2	6			6					
16 ..	39	25	1	5		7		1						
20 ..	55	32		10	3	2	2		1					5
23 ..	55	45		4	2			1	1					2
27 ..	81	65		10	4	1								1
30 ..	12	11		1										
December 4 ..	12	11					1							
7 ..	29	24	1	3										1
11 ..	27	20	1	3		2	1							
15 ..	25	19				2								4
18 ..	8	7												1
21 ..	13	11	1			1								
27 ..	43	13		13	1	5								11
28 ..	136	29	20	79	1									7
1934—														
January 2 ..	19	6		7		1								5
4 ..	55	15	3	23		2								12
9 ..	13	5		2		1								5
11 ..	51	33		14		1								3
15 ..	21	10		10			1							
18 ..	59	16	5	21	4	5								6
22 ..	89	27	6	43			10							3
25 ..	18	13		5										
30 ..	31	17	4	7			1							2
February 1 ..	15	13					2							
5 ..	25	19	3	3										
8 ..	69	32	5	23			1					2		6
12 ..	27	22		5										
16 ..	35	31		4										
19 ..	5	1		2			1							1
22 ..	17	12		2								3		
26 ..	58	31	10	6										11
March 1 ..	8	7		1										
5 ..	22	19	3											
9 ..	19	6	1	12										
14 ..	26	18		1								5		2
20 ..	10	10												
22 ..	10	9		1										
26 ..	2	2												
28 ..	30	28	2											
April 4	7	7												
10	3	1										2		

amounts. Whether this is due to their not being frequent in the district or to their high specific gravity causing them to fall has not been ascertained up to the present. The provision of wings on the part of the pine pollen may be required by its high specific gravity, but they do not appear to have allowed the pollen to reach the station to any great extent.

The pollens of eucalyptus and acacia were only rarely found and therefore have not been included in the chart. Apparently they were not borne very far by the wind.

Various composite pollens were found occasionally throughout the whole period and especially during January. They were not differentiated and no graph was made of their occurrence.

The types and quantities of pollen collected are shown in the accompanying table.

Summary.

A survey was made of the amount of pollen present in the air around Melbourne during the hay fever season of August, 1933, to March, 1934. The method used was similar to that used in America by Scheppegrell and later by others. The commoner types of pollen were distinguished and graphs made of the frequency of their occurrence, so that some idea of their pollination period was obtained.

An attempt was made also to correlate the amount of pollen present in the atmosphere with the amount of rainfall, the percentage of relative humidity, and the quantity of northerly winds operating. It was found that the rain caused the pollen to fall out of the atmosphere and was the most potent single climatic influence which was investigated. Northerly winds were usually associated with a high pollen count. The humidity had apparently little influence, but unfortunately it was not possible to make observations every day. It is hoped that the survey for this season will be more complete. Observations are being made every day and the technique is improved.

References.

(1) C. H. Blackley: "Experimental Researches on the Cause and Nature of Hay Fever", 1873.
(2) Dunbar: *"Zur Ursache und specifischen Heilung des Heufiebers"*, 1903.
(3) L. Noon: "Prophylactic Inoculation against Hay Fever", *The Lancet*, Volume I, 1911, page 1572.
(4) J. Freeman: "Further Observations on the Treatment of Hay Fever: Hypodermic Inoculations of Pollen Vaccines", *The Lancet*, Volume II, 1911, page 814.
(5) W. Scheppegrell: "Hay Fever and Hay Fever Pollens", *Archives of Internal Medicine*, Volume XXV, 1917, page 959; "Hay Fever and its Relation to 100 of the Most Common Plants, Trees and Grasses", *Medical Record*, Volume XCII, 1917, page 230; "Seasons, Causes, Geographical Distribution of Hay Fever Plants and Hay Fever Resorts in U.S.A.", *Public Health Reports*, Volume XXXV, 1920, page 2241. "Analysis of Six Annual Seasons of Fall Hay Fever in New Orleans", *Public Health Reports*, Volume XXXVIII, 1923, page 1565; "Hay Fever and Asthma: Cause, Prevention and Cure", 1922; "Airplane Tests of Hay Fever Pollen Density in the Upper Air", *Medical Journal and Record*, Volume CXIX, 1924, page 185; "Immunological Classification of Common Hay Fever Plants and Trees", *New York Medical Journal*, Volume CXVII, Number 12, 1923.
(6) P. Acquarone and L. N. Gay: "Pollen Flora in Baltimore during 1929", *Journal of Allergy*, Volume II, 1930, page 337.
(7) H. K. Detweiter and Helena Hurst: "Studies on the Pollen Content of the Air, Toronto, Canada", *Journal of Allergy*, Volume I, 1929, page 334.
(8) W. W. Duke: "Dropping Out of Pollen Seasons", *Journal of Allergy*, Volume II, 1930; "A Rapid and More Accurate Method of Determining the Pollen Content of the Air", *The Journal of the American Medical Association*, Volume XCIX, 1932, page 1687.
(9) W. W. Duke and O. C. Durham: "Pollen Content of the Air", *The Journal of the American Medical Association*, Volume XC, 1928, page 1531.
(10) O. C. Durham: "Typical Fall Surveys of Hay Fever Flora", *Journal of Laboratory and Clinical Medicine*, Volume X, 1924, page 610; "The Contribution of Air Analysis to the Study of Allergy", *Journal of Laboratory and Clinical Medicine*, Volume XIII, 1927, page 967; "Incidence of Ragweed Pollen in U.S.A. during 1929", *The Journal of the American Medical Association*, Colume XCIV, 1930, page 1907; "Comparative Studies in Ragweed Pollen Incidence: Atmospheric Data from 22 Cities", *Journal of Allergy*, Volume I, 1929, page 12; "The Ragweed Season of 1931", *Journal of Allergy*, Volume III, 1932, page 357; "The Ragweed Season of 1932", *Journal of Allergy*, Volume IV, 1933, page 105.
(11) L. N. Gay: "Pollen Flora in Baltimore during 1929", *Journal of Allergy*, Volume II, 1930, page 337.
(12) H. M. Hall: "Hay Fever Plants of California", *Public Health Reports*, Volume XXXVII, 1922, page 803.
(13) J. P. Henry and A. L. Herring: "A Botanical Survey of Memphis, Tennessee and Surrounding Territory", *Journal of Allergy*, Volume I, 1929, page 163.
(14) I. S. Kahn: "Botany of S.W. Texas with Reference to Hay Fever and Asthma", *The Journal of the American Medical Association*, Volume LXXXII, 1924, page 871.
(15) K. K. Koessler and O. C. Durham: "A System for an Intensive Pollen Survey", *The Journal of the American Medical Association*, Volume LXXXVI, 1926, page 1204.
(16) P. M. Patterson and O. C. Durham: "Pollen Content of the Air and its Relation to Hay Fever in Baltimore, Maryland", *Journal of Allergy*, Volume III, 1931, page 3.
(17) W. T. Penfound and B. G. Efron: "Herbaceous Plants in New Orleans in Relation to Allergy", *Journal of Allergy*, Volume I, 1929, page 369; "Standardized Method for Pollen Air Analysis", *Proceedings of the Society of Experimental Biology and Medicine*, Volume XXVII, 1930, page 650.
(18) E. W. Phillips: "Pollen Incidence in Central Arizona", *Journal of Allergy*, Volume III, 1931, page 489.
(19) F. M. Rackeman and L. B. Smith: "The Plant Pollens of New England: Their Relative Importance", *Journal of Allergy*, Volume II, 1930, page 364.
(20) A. H. Rowe: "A Study of the Atmospheric Pollen and Botanic Flora of the East Shore of San Francisco Bay", *Journal of Laboratory and Clinical Medicine*, Volume XIII, 1927, page 416; "Botanical Survey of San Joaquin Country in Central California", *Journal of Allergy*, Volume III, 1931, page 375.
(21) R. F. E. Stier and G. Hollister: "A Botanical Survey of Hay Fever-Producing Plants in the Pacific, N.W.", *Journal of Allergy*, Volume I, 1929, page 260.
(22) R. P. Wodehouse: "Atmospheric Pollen", *Journal of Allergy*, Volume IV, 1932, page 220.
(23) L. A. Ivan Maxwell: "Hay Fever and Asthma", The Medical Journal of Australia, Volume I, 1923, page 693.
(24) E. Derrick: "Notes on the Causes of Hay Fever and Asthma in Australia", The Medical Journal of Australia, Volume II, 1929, page 603.
(25) G. T. Moore and V. L. La Garde: "The Identification of Pollen from So-Called Hay Fever Plants", *Annals of the Missouri Botanical Gardens*, Volume XIII, 1926, page 355.
(26) R. P. Wodehouse: "The Phylogenetic Value of Pollen Grain Characters", *Annals of Botany*, Volume XLII, 1928, page 892.

SOME OBSERVATIONS ON THE ÆTIOLOGY OF GENERAL PARALYSIS OF THE INSANE.

By C. R. D. Brothers, M.D., B.S. (Melbourne),
Assistant Medical Officer, Mont Park Mental Hospital.

Although it has now been definitely established that the essential cause of general paralysis is infection with the germs of syphilis, and although general paralysis is no longer regarded as a metasyphilitic or parasyphilitic disease, that is, as something different from an active syphilitic infection, there are still so many obscure points about its ætiology that it seemed worth while to record some pertinent personal observations made during the past few years while engaged in the malarial treatment of neuro-syphilitics at Mont Park. The following facts have been gathered from the histories of one hundred male general paralytics admitted consecutively to the Mont Park Mental Hospital during the latter half of 1932, 1933 and 1934. Juvenile paralytics have been excluded.

The average age of the patients on admission was found to be 45·2 years. There were 50% whose ages were between 40 and 50; 23% were over 50, and of these nine were over 60, the eldest patient being 67; the remaining 27% were under 40, and of these two only were below the age of 30, the youngest being 28. In this series 52% gave a history of having acquired syphilis, the average age at the time of infection

being 28·1. The remaining 48% denied all knowledge of ever having had the complaint, although in the majority of cases they would have had no compunction in making such an admission.

It is difficult to estimate the exact period of incubation, as the onset of the symptoms of general paralysis is so frequently insidious, and even in acute cases a careful inquiry will often elicit a history of vague personality changes or aberrations of behaviour over the preceding years. For convenience we have taken the incubation period to mean the time elapsing between infection and admission to hospital. In our series the average period was 14·1 years, the extremes being 4 and 31 years respectively. In 40% of cases the incubation period was between 10 and 15 years, with 36·5% over 15 and 23·5% below 10.

As only 2% to 4% of syphilitics eventually become affected by general paralysis, it is clear that there must be some other factor in addition to syphilitic infection which determines the development of the disease: whether this factor depends on a special strain of the organism or whether it is due to individual susceptibility. The only evidence we have to offer in support of the theory that a special strain of spirochæte is responsible is a single instance in which a man and his wife were admitted for treatment within a few months of each other, both having contracted syphilis ten years previously, presumably the one having infected the other.

Admittedly a psychopathic heredity can be found in quite a number of general paralytics, but whether or not it plays any part in the actual causation of their disease is open to question. Bolton states that he found evidence of psychopathic heredity in as many as 81·9% of his cases. He goes on to say: "The subjects of this form of mental disorder would, if they had not been syphilized, have suffered from one or other form of neuronic dementia." In some 15% of our own cases we elicited a history of psychoses occurring in near relatives—parents, brothers or sisters. Of special interest are four cases in each of which there is a family history of general paralysis. In one case both the father and one brother of the patient died of the disease a number of years ago. In the second the patient has a sister who is at present an inmate of Mont Park, suffering from general paralysis. In the third and fourth instances the father of one and the uncle of the other both died of general paralysis. These cases would surely indicate some familial susceptibility. There was no reason in any of these four cases to suspect juvenile paralysis. The patients were all approaching forty or over on admission and showed none of the stigmata of congenital syphilis.

At least another 11% could be defined as "poor types", consisting originally of high grade mental defectives or neuropaths. Two patients had had mental breakdowns prior to contracting syphilis. One had had an attack of *dementia præcox*, and the other an attack of melancholia. Both were clinically and serologically general paralytics on their last admission. Three other patients had been from an early age sexual inverts, and all three of them gave a history of extragenital chancres. It is of interest to note that each of the three passed into a state of "galloping" general paralysis of the insane and died shortly after the termination of treatment with malaria.

Summing up, it would appear that 29% of patients showed predisposing constitutional defect. This is a conservative estimate, as in many cases no reliable family history was available.

Of the 9% who were over the age of sixty, the majority were provisionally labelled senile dements, the diagnosis being based on their mental condition alone. However, a routine clinical and serological examination subsequently proved them to be general paralytics. Possibly the condition of these patients could have been explained by assuming that in each case general paralysis had supervened on an already deteriorating neuronic structure, but whether in the absence of syphilis they would have developed a senile psychosis is difficult to state with any degree of certainty.

Mental strain has often been regarded as predisposing to the development of general paralysis, but the influence of this factor is difficult to assess. It has been held in the past that active, hard-working business men are especially liable to develop general paralysis after syphilitic infection. In the present series only 7% could be styled as business men and they were nearly all "small" business people. The complete figures, embracing all occupations, are as follows: unskilled labourers 49%, skilled labourers 35%, clerks 7%, business men 7%, and musicians 2%. There were no professional men. The returned soldiers accounted for 23%.

As there is a remarkable similarity between the signs and symptoms of certain alcoholic psychoses and those which result from syphilitic disease of the brain, it seems only reasonable to expect that when both toxins of alcohol and syphilis are acting simultaneously on the same nervous system, the one will reinforce the other, thus hastening the degenerative process. Whilst not denying the importance of the influence of alcohol in the ætiology of general paralysis, it must be remembered that quite a number of tertiary syphilitics attending our general hospitals or private practitioners have been more or less heavy alcoholics and yet have not developed general paralysis. No less than 24% of our patients gave a history of excessive alcoholism over a period of many years. Those cases in which alcoholism appeared as a symptom of developing general paralysis have, of course, been excluded. In one striking case the patient had quite a long list of police court convictions and had been admitted on no less than five occasions to another mental institution during the last twenty years, on each occasion suffering from acute alcoholic insanity. Only two of the twenty-four alcoholics were recognized methylated spirit drinkers. During the past three years the average number of male general

paralytics admitted to Mont Park each year was 45. Compared with 1912-1915, when the average number admitted to Victorian mental hospitals was 63, this is a reduction of 29% in the incidence of general paralysis. This might be explained by either a lower incidence of acquired primary syphilis or by more effective and intensive antispecific treatment during the early stages of the disease. Some neurologists, however, claim that parasyphilitic disease of the nervous system has shown a relative increase in frequency since the advent of arsenical compounds in the treatment of primary and secondary syphilis. Whilst 28% of our patients were treated with "Salvarsan", bismuth *et cetera* during the early stages, the majority admittedly received most inadequate treatment, often consisting of one course of five or six injections. However, 8%, received intensive treatment over many months, and in one case for three years, and all were given to understand they were cured. Yet ten years or so later they were admitted to a mental hospital with general paralysis. Incidentally, not a single patient in our series had ever had his cerebro-spinal fluid examined whilst in the early stages of syphilis. Probably had the cerebro-spinal fluid of all the patients been examined at that stage they would have yielded positive Wassermann reactions, indicating in each case a still active syphilitic process, even if no reactions had been given by the serum at the same time.

Acknowledgement.

I am indebted to Dr. Catarinich, Medical Superintendent of the Mont Park Mental Hospital, for permission to publish the above clinical material.

THE PROBLEMS OF ANTENATAL CARE.

By T. Dixon Hughes, M.B., Ch.M., F.R.A.C.S.,
Honorary Assistant Medical Officer, Women's Hospital, Crown Street, Sydney; Tutor in Obstetrics, University of Sydney.

There has arisen in England a controversy as to the value of antenatal care because the maternal death rate has shown no improvement since its introduction. Before passing judgement on such a vital subject it would be well to examine the state of affairs as they exist there today. A great deal of the antenatal care is carried out by part or whole-time medical officers in antenatal clinics. These officers are not concerned with actual delivery and therefore are divorced from the opportunity of studying the many vicissitudes which beset the patient in labour, and of checking their prognosis as to the type of labour.

The English authorities are deeply concerned with this lack of coordination between the antenatal clinics and the medical staffs and practitioners of the surrounding districts. This is a matter that we in Australia must consider, because there is a belief abroad among some people that the establishment of antenatal clinics somewhat on the lines of baby clinics would do much to lower the maternal death rate.

Antenatal care can do this, and will, if it is carried out on proper lines, but to follow the present English custom will but bring discredit to what is one of the great advances in preventive medicine. This is best illustrated by the following figures. The death rate among 5,528 patients who attended at least once at the Women's Hospital, Crown Street, Sydney, for antenatal care during 1931 to 1934 was 2·91 per thousand, compared with a death rate of 4·88 per thousand for the State of New South Wales for the ten-year period 1921-1930.

Figures from certain hospitals set out in the accompanying table further demonstrate this fact.

In comparing the figures from the Women's Hospital, Crown Street, with those of the first three hospitals on the list, it must be realized that the present definition of a booked case at the Women's Hospital is that the patient shall have attended at least once at the out-patients' department. In the first three hospitals mentioned in the table, however, a more rigid control is kept over the patients. The hospitals insist upon a much higher standard of attendance and the patients are visited in their homes if they fail to attend.

All these institutions are deeply concerned with the responsibility of the delivery of the patient and they are in the position to consider the antenatal treatment in the light of results. The most complete antenatal service will break down in the face of

Table to Show How Degree and Character of Antenatal Care Affect Mortality in Pregnancy and Childbirth.[1]

Institutions.	Category A ("Booked").			Category B ("Emergencies").		
	Total Cases.	Deaths.	Percentage.	Total Cases.	Deaths.	Percentage.
General Lying-in Hospital, York Road (1928-1931)	4,051	3	0·07	150	2	1·33
East End Maternity Hospital (1928-1931)	5,448	7	0·12	59	3	5·80
Queen Charlotte's Maternity Hospital (1926-1930)	9,836	27	0·27	933	49	5·25
Women's Hospital, Crown Street, Sydney (1931-1934)	5,528	17	0·29	1,007	21	2·09
Liverpool Maternity Hospital (1926-1930)	6,111	33	0·49	1,542	63	4·09
Simpson Memorial Hospital, Edinburgh (1929-1931)	3,690	24	0·65	2,393	82	3·42
Glasgow Royal Maternity Hospital (1926-1930)	7,995	102	1·20	11,656	424	3·60

[1] This table is modified from "Maternal Mortality and Morbidity", by J. Munro Kerr.

mismanagement at the time of delivery. Therefore antenatal care and the delivery of the patient are undoubtedly bound up with one another, and in ordinary circumstances the best person to carry out the antenatal care is the person who is going to be responsible for the delivery. Apart from the indigent poor, for whom only the clinic should cater, this should be the patient's own doctor. The private medical attendant is best suited to carry out antenatal care by virtue of his personal knowledge of the patient, her habits and temperament, and of his ability to create an atmosphere of safety and security and to prevent fear, the cause of many inertias. To render this service he must so equip himself that the need for multiple clinics will not arise. It is well at this stage that we should examine what antenatal care entails.

Antenatal care is preventive medicine and not a doctrine of unwarranted interference based on false or inexperienced judgement. If antenatal care means increased operative interference, it destroys its very aim and will retard the onward march that must ensue from its wise application. Antenatal care has been defined as the expectant watchfulness of the expectant mother. It is not the mere taking of a few measurements and testing the urine from time to time. It entails a complete medical and family history, together with a thorough examination of the patient. By this means the medical practitioner is able to establish a normal standard for the particular patient and thus to evaluate with more precision any deviation from this standard at a later date. Not the least important function of the attendant throughout the whole period of care is to create confidence in the patient's mind and abolish the fear that is instilled into so many *primiparæ* by their kind friends; for it is a well known fact that the highly strung or nervous patient is more prone to inertia.

The aim of antenatal care is: (i) to reduce the maternal mortality and morbidity; (ii) to reduce the natal and neonatal death rate. It appears that the gradual improvement in the latter is being obtained partly at the expense of the former.

It is but logical that if toxæmias are treated, malpresentations noted, and the obstetrician forewarned of impending trouble, the maternal death rate should show some improvement. Why has it failed to do so? The answer must be that: (i) a sufficient proportion of mothers have not availed themselves of antenatal care through apathy or ignorance; (ii) antenatal care has not been efficiently carried out; (iii) antenatal care has increased the operative interference rate, and by so doing has counteracted the benefit that it has conferred by its other activities.

Is it not possible that in an attempt to reduce obstetrics to a mathematical problem unnecessary interference has taken place, in the form of an increase in the number of Cæsarean sections and inductions of premature labour? That this is a possibility must be brought home to anyone concerned with antenatal care. Such a person is inundated by the number of patients who appear with the suggestion that in view of the small pelvic measurements some active measures should be taken; and a large proportion of these patients deliver themselves of a normal, healthy offspring with surprising rapidity. It is therefore fitting to consider whether the induction of premature labour and Cæsarean section in *primiparæ* has any place in obstetrics when from antenatal examination a normal full-time labour is considered impossible. The factors that might lead to such conclusions are: (i) small pelvic measurements, (ii) some degree of disproportion between the fœtal skull and pelvis.

Let us consider these factors in more detail.

First, in regard to small pelvic measurements, if the finding of small pelvic measurements, without regard to whether the fœtal head can be made to fit into the pelvis, is going to lead a practitioner to recommend induction of premature labour, it would be better, as Dr. Aleck Bourne[2] suggests, to "abolish the pelvimeter". That it does so influence the attendant will be borne out by all those in contact with antenatal work. Dr. W. S. A. Griffith,[3] senior consulting surgeon, Queen Charlotte's Maternity Hospital, London, says, as the result of the examination of series of pelves, that "while external measurements give valuable information in indicating the type and some indication of the degree of deformity, they do not, except in severe cases, enable him to determine accurately the internal measurements and certainly not to decide on such grave issues as Cæsarean section or even induction of labour". This is a clear and concise statement of the position that pelvimetry should play. Unfortunately the love of mathematical formulæ has led many throughout the world to believe in its infallibility, even to the extent of advising against marriage.[1]

Secondly, where some degree of disproportion between the fœtal skull and the pelvis is found antenatally, is surgical induction of premature labour or Cæsarean section in *primiparæ* advisable?

Let us consider the question of surgical induction of labour.

The theory of induction before term as a means of overcoming disproportion is an attractive one. It presupposes a normal delivery of a premature child through passages too small to allow the birth of a normal infant. On what exact evidence can it be said that the passages will not allow the delivery of a normal child? We have previously shown that a diagnosis based solely on the external measurements of the pelvis is most unreliable. It is the relationship of the size of the fœtal head to the maternal pelvis that is the main problem. Theoretically, a disproportion should be a sound indication for induction of premature labour. There are, however, but few who can with certainty predict that a normal delivery will be impossible at term, but

[1] A patient who came under the writer's own observation and for whom marriage was not considered wise on the above grounds ultimately had a normal infant without forceps.

can be carried out with safety to the mother and child at about eight months. Induction of labour in itself is not without risk to the mother and child, and the apparent logical simplicity of induction of premature labour is a snare and a delusion. It is fraught with anxiety and uncertainty as to the action on the uterus and the result to the child. De Lee[4] gives the maternal mortality rate as 1·4% and the fœtal mortality from 30% to 60%. And Johnston,[5] in speaking of induction as a method of treatment of pelvic contraction, says:

> In a first pregnancy the operation should not be done, as a first labour is always somewhat of an experiment and the child must be given the benefit of the doubt.

Further, Whitridge Williams[6] states that from 70% to 80% of all labours complicated by contracted pelvis will end spontaneously if left alone; and he holds that the induction of premature labour is no longer justified. De Lee,[7] referring to various complications that may arise following induction of labour, says:

> All these complications have been observed by the author and have much reduced his enthusiasm for the induction of premature labour in contracted pelvis, and he rarely performs it.

E. J. Browne[8] remarks that induction of premature labour and Cæsarean section at term for disproportion are good examples of unnecessary intervention in antenatal care.

Another danger with mechanical induction is that the head may fail to descend, and then the possibility of a Cæsarean section with any degree of safety has been removed; further, should forceps have to be applied to the premature infant, there is an added element of danger, as the work of Holland has shown that there is an increased risk of cerebral injury associated with prematurity. And as a result of observing some thousands of patients at the Women's Hospital, Crown Street, the writer has come to the very definite opinion that surgical induction of premature labour in *primiparæ* for disproportion has no part in antenatal care. Also Cæsarean section in *primiparæ*, in the vast majority of cases, should not be performed without some trial of labour.

What has been said concerning the indications for the induction of premature labour applies equally to the performance of Cæsarean sections in *primiparæ* without some trial of labour. Here the mortality rate is reversed, the maternal death rate being high and the fœtal death rate being low. Although the operator is liable to forget his failures, the mortality rate cannot be kept below 2%, compared with 0·4% for delivery *per vias naturales*.

Cæsarean section is an easy way out of any difficulty, and it is but human to take the easy road which, after a succession of a few cases, seems a safe one. There is, however, that inevitable pitfall of 2% maternal mortality. Cæsarean section is dramatic; it appeals to the lay mind; it brings greater recognition to the attendant than the wise guidance of a normal labour.

Let the keynote be one of restraint, tempered with the wisdom of timely interference, and let the labour be conducted so that wise conservatism be not turned into foolish obstinacy. Then will Cæsarean section be done after trial of labour and not after an exhaustion by labour; so will it come into its proper sphere.

References.

(1) J. M. Munro Kerr: "A Study of the Problem of Maternal Mortality and Morbidity", page 193.
(2) Aleck Bourne: "A Plea for Abolition of the Pelvimeter", *The British Medical Journal*, November 24, 1934, page 963.
(3) Walter S. A. Griffith: "A Plea for the Abolition of the Pelvimeter", *The British Medical Journal*, December 8, 1934, page 1075.
(4) De Lee: "Principles and Practice of Obstetrics", Fifth Edition, page 1101.
(5) R. W. Johnstone: "Text Book of Midwifery", Second Edition, page 323.
(6) J. Whitridge Williams: "Obstetrics", Fifth Edition, page 425.
(7) De Lee: *Loco citato*, page 761.
(8) E. J. Brown: "Are We Satisfied with the Results of Ante-Natal Care?" *The British Medical Journal*, August 4, 1934, page 194.

THROMBOPHLEBITIS MIGRANS, WITH AN ILLUSTRATIVE CASE.

By Mostyn L. Powell, M.B., M.R.C.P.,
Honorary Physician to Out-Patients, Prince Henry's Hospital, Melbourne; Associate Assistant Physician, Melbourne Hospital.

Thrombophlebitis migrans is distinctly a rare disease. Its interest therefore may appear academic only, but I feel that the comments culled from the case herein described, and from its predecessors in the rather scanty literature, are worthy of publication for the following reasons:

1. The syndrome is not a well recognized one.
2. Its recognition or otherwise makes a tremendous difference in regard to the management and prognosis and, incidentally, to the peace of mind of the man in charge.
3. It is possible, too, that the disease is not so rare as it appears; many may recall similar cases in which a diagnosis was not made, but recovery occurred.
4. The characteristics which give the disease its name make it of interest to most branches of the profession, even, as this case shall show, to the surgeon, albeit in a somewhat negative way.

Case History.

Miss S., aged fifty-one years, a rubber worker (whose duties brought her into contact with rubber and rubber adhering solution only), was in good health until early in October, 1934, when she began to feel vaguely off colour. At this time and at intervals of about three days she developed acutely tender and painful areas in the left thigh, right calf and right heel. Nothing was visible, but when she touched these areas or endeavoured to walk, they were exquisitely tender.

Five days later the left groin became painful and there appeared a tender, egg-shaped swelling, which disappeared in about three or four days. During the last week in October she developed a painful, tender lump, which she herself could feel, in the left side of the lower part of the abdomen. This gave her great pain and she complained of "a grating feeling as I breathed".

Gradually, over a period of two weeks, this subsided, but before her abdominal pain was quite better, on November 2, 1934, the next epoch began in the form of acute pain, aggravated by breathing, in the right side of the lower part of the chest. There was very little cough, no sputum, and in five days her chest pain had gone, leaving "only a few clicks", and though weak and her right calf still tender, she felt fairly well. It was at this stage that I was asked to see her.

Her past history showed little of importance. Six years before she had had pyorrhœa and all her teeth were then extracted. In April, 1934, she had had influenza, with some cough and sputum, but had recovered apparently perfectly. There was no history of a sore throat or any infective process immediately prior to her illness. She was subject to migraine. Her family history contained nothing of importance.

On November 8, 1934, one month after her first symptoms, examination revealed the following points. She was a pale, sick, rather obese woman. Her temperature was 38·3° C. (101° F.), her pulse rate was 120 and her respiratory rate 30 per minute. Her systolic blood pressure was 160 and her diastolic pressure 100 millimetres of mercury. Her eyes, including the fundi, appeared normal. Her tongue was dry and furred. She was edentulous. The throat was reddened, but clear.

Apart from a faint systolic bruit and a rather booming first sound at the apex, there was no abnormality detected in the heart. Examination of the lungs showed the right base to be dull posteriorly; there were many moist sounds here, and a friction rub was plainly heard. There were a few moist sounds also at the left base posteriorly.

The abdomen was full, but moved fairly well with respiration. Neither liver nor spleen was palpable. In the lower part of the abdomen, lying transversely, more in the left side, was a smooth, firm, tender mass, not moving with the limited respiratory excursus available. The inguinal regions were now absolutely clear.

The right calf and heel were very tender, but there was nothing definitely palpable. There was a tender spot on the outer aspect of the left thigh, and a slight but definite pitting œdema over both lower tibiæ was present.

Examination of the nervous system revealed no abnormality and nothing abnormal was found on vaginal examination.

A blood examination showed the following points:

Red blood cells, per cubic millimetre ..	4,164,000
Hæmoglobin value (Tallquist) ..	70%
White blood cells, per cubic millimetre ..	11,000

A film showed the leucocytosis to be of a mature polynuclear variety only. The red cells were normal. The urine showed no abnormality.

Discussion.

As is so often the case in medicine, the history was the most important clinical finding. The sequence of sudden onset of pain in localized regions in the inferior extremities, the groin, abdomen and chest was surely vascular, and reminiscent of a pyæmia or of embolic phenomena from some focus such as an ulcerative endocarditis or a septic uterus. Yet these phenomena were, apart from their pain and discomfort, singularly innocuous, and those in the left groin so completely so that the patient's word was the only evidence of its ever having occurred. Moreover, the patient, though admittedly pale, inert and definitely ill, did not give the impression of profound toxæmia and impending death, usually associated with the diseases mentioned above.

On consideration of these points the site of the lesions was judged to be in the venous side of the circulatory tree; and the whole case fitting very closely the syndrome described with four illustrative cases by Moorhead and Abrahamson[1] in 1928 and by Ryle,[2] with five cases, in 1930, and termed *thrombophlebitis migrans*.

General treatment was instituted, particularly towards relief of the pain; the patient was told of the possibility of further attacks, but that she would ultimately recover. The relatives were warned of the possibility of sudden hemiplegia or monoplegia, the benign and rapidly recoverable nature of such being stressed.

Subsequently the patient did develop further thromboses in the upper extremities; a little fluid collected at the base of her right lung, but was not removed. She now (February, 1935) is slowly getting about and definitely improving. She still has a mild, persistent œdema of the ankles and is slightly anæmic.

Thrombophlebitis migrans may be defined in a comprehensive, if somewhat cumbersome way, as a benign disease of adult life, of obscure, but probably infective, origin, running a variable course of about four months to five years, characterized by recurrent, fever-accompanied bouts of inflammation and thrombosis in short lengths of medium and smaller sized veins anywhere in the venous tree, severe pain being a constant feature (except in the cerebral lesions), but the other accompanying symptoms and signs depending on the region affected.

As in this case, the lesions usually appear first in the extremities and later in the viscera, but this is by no means a rule; and intraabdominal and thoracic lesions have on occasions ushered in thromboses in the extremities. It may well be asked why these are not embolic in many cases from an original thrombosis.

But the onset of the lesions and their subsequent course are so unlike those of emboli of sufficient size to produce pleural friction, pleural effusion and pulmonary infarction or large, easily palpable abdominal tumours. Such emboli have a cataclysmic onset—often death rapidly follows in the pulmonary cases; rapid ileus follows in the abdominal emboli. The obvious explanation is that the lesions of *thrombophlebitis migrans* are venous. Embolic phenomena are in most cases arterial.

Also, though the lesions of *thrombophlebitis migrans* are definitely of rapid onset, there is often some preliminary warning in the way of some pain and tenderness in the region—almost an aura. As a rule, large emboli give little warning. Also the interval between events often amounts to many weeks, which is rather long for a typical case of multiple embolism.

Of the deep lesions of this strange disease the three common ones are lung, mesenteric or omental, and cerebral, with rarely, and somewhat open to doubt, coronary. Even in such vital regions as these recovery is the rule and can be confidently predicted.

Thus the lung lesion typically produces a pleurisy, some underlying consolidation, occasionally a small sterile effusion, but ultimate recovery. One of Ryle's cases went further and produced offensive sputum,

suggesting a breaking down infarct, before complete recovery occurred.

If the lesions are omental, as I suspect was the case in the present patient, there is acute abdominal pain, the formation of a mass of engorged omentum, an aseptic localized peritonitis, adherence to surrounding viscera, but again recovery occurs. Even if the lesions are mesenteric, all that happens is that the engorged vessels in the wall of the gut give way, producing a mild melæna, but the bowel segment does not die, collateral vessels take over the circulation, and recovery ensues. How different from the arterial block of an embolus! Incidentally it will be apparent that a surgical opinion may well be requested in these cases, particularly if an intra-abdominal lesion ushers in the disease.

If a cerebral vein be involved, the result will obviously depend on the region involved. There is usually a good deal of headache, perhaps a sudden hemiplegia or monoplegia or, as in a case reported by Campbell and Morgan,[3] in which the occipital circulation was involved, sudden development of tubular vision taking place, again with complete recovery in several days.

The occurrence of a coronary venous thrombosis in this disease is very rare. In one of Moorhead and Abrahamson's cases there was, during a typical series of thrombotic episodes, a sudden bout of anginal pain accompanied by a rapid fibrillation, which cleared up in an unstated period. Legrand[4] records a similar event in a case of his.

Carey Coombs,[5] in his article on the relation of anginal pain to anoxæmia of cardiac muscle, mentions a series of five cases in which there were sudden attacks of apparent cardiac infarction, associated with thrombophlebitis elsewhere, and in which excellent recoveries followed, but he makes no claim for these cases to be labelled *thrombophlebitis migrans*. In the one case which he details, the phenomena began in a man in perfect health six weeks after a fractured fibula, with pain in the chest and hæmoptysis. Ten days later a gangrenous appendix was removed, and a fortnight later he had his cardiac episode, with a sense of oppression in the chest, rapid pulse, and a pericardial friction rub. A week later he had his final lesion—a severe thrombophlebitis extending down from the left groin. It will be seen that there are several points here to cast doubt on a diagnosis of *thrombophlebitis migrans*, at any rate within the specific meaning of the term, but it points the truism that if a coronary thrombosis is venous, as presumably this was, then excellent recovery may ensue. It is not inconceivable that some of the coronary cataclysms which do so unexpectedly well occasionally are venous and not arterial in origin.

These, then, are the highlights of the disease. In the background is a chronic state of ill-health, with weakness, lassitude, anorexia, dyspnœa on exertion, and pallor as definite features. A clearing of this general state is often the signal for a welcome cessation of the local lesions, but the latter often leave in their train mild reminders of their activities, in the form of slight pitting œdema of the extremities or a thickened pleura which aches its way into consciousness each winter.

Ætiology.

It is difficult to escape the conviction that this disease is infective in nature, that the organism is blood-borne, with its particular seat of operation the walls of the venous channels of the body. The polymorphonuclear leucocytosis and secondary anæmia, too, are suggestive, though the former, of course, could be the aftermath of a thrombosis, non-infective in origin. The vague background of chronic ill-health mentioned before is also strongly suggestive of a circulating toxin, with or without the organism producing it. Legrand did actually isolate a streptococcus on two occasions, but neither Moorhead and Abrahamson nor Ryle was able to confirm this. Septic teeth have been invoked as a possible source, but the patient under discussion had been edentulous for seven years. (It is always possible, of course, for an edentulous patient to have retained infected roots.) Severe *pyorrhœa alveolaris* was found in one of Moorhead and Abrahamson's patients, but the remainder had no evidence of a septic focus anywhere in the body. Syphilis, tuberculosis and gout have been mentioned as possible ætiological factors, but there is certainly no evidence of any constant relation. It should be mentioned that Buerger[5] states that some cases of *thromboangiitis obliterans* have been preceded by wandering venous thromboses, but it is very obvious that the vast majority of both diseases occur without such relation.

Summary.

1. A case typical of the disease known as *thrombophlebitis migrans* is described.

2. The disease is defined and discussed and its favourable prognosis emphasized.

3. It is suggested that the condition is commoner than is realized and that many may recall similar cases which have passed into memory as interesting cases, undiagnosed, but in which recovery occurred.

4. The views of earlier writers on its probable infective origin are supported.

5. Although no specific therapy can be suggested, the patient can at least be made comfortable in mind and body, and it is perhaps something to be able to say with conviction: "This man has *thrombophlebitis migrans* and he will get better."

Acknowledgement.

I am indebted to Dr. Roy L. Park for permission to use this case as a basis for this article and for his help in the observations of the various episodes.

References.

(1) T. G. Moorhead and L. Abrahamson: "Thrombo-Phlebitis Migrans", *The British Medical Journal*, April 7, 1928, page 586.
(2) J. A. Ryle: "Thrombo-Phlebitis Migrans", *The Lancet*, October 4, 1930, page 731.
(3) M. Campbell and O. G. Morgan: "An Unusual Case of Multiple Thrombosis", *Guy's Hospital Reports*, Volume LXXX, 1930, page 34.
(4) C. Legrand: *"La septicémie veineuse subaiguë"*, *Revue de Médecine*, 1925, Volume XIII, page 169.
(5) Buerger: "The Circulatory Diseases of the Extremities", 1924, page 279.

Reports of Cases.

A RETROCAECAL APPENDIX.

By J. L. MEAGHER, M.B., B.S.,
Marist Mission, North Solomons.

IT is well known that the retrocaecal appendix presents anomalous symptomatology. A case of the kind presenting some unusual features, occurring in the practice of the writer at Buka Passage, North Solomons, is herewith cited.

The patient was an infant welfare nurse attached to the staff of the Marist Mission in the North Solomons. She was an active, healthy woman of twenty-four, without history of previous illness at the time of the first attack of abdominal pain in July, 1934. Throbbing pain was felt in the right side of the lower part of the abdomen, and the patient was obliged to remain in bed three days. Vomiting, self-induced, gave relief. The second attack occurred suddenly one month later—one Sunday morning about 9 o'clock, and compelled the patient to leave a religious service. She vomited, and within half an hour the pain was completely relieved. The pain was felt in the right pelvic region and was agonizing. There was a history of a dietetic indiscretion on the evening before this attack. At intervals during the next two months a feeling of discomfort and soreness in the region was present at times, but the patient nevertheless continued at her nursing work.

The third attack occurred on October 7 and again sharp pain was felt. The patient was attended for the condition by the writer for the first time on October 7, on the day after the onset of pain. Premonitory symptoms lasting two or three days had been nausea, insomnia and a slight looseness of the bowels. Upon examination there was manifest generalized tenderness on the right side of the abdomen, with some para-umbilical soreness and moderate but definite guarding of the muscles in the lower right pelvic region. A definite but lesser degree of sensitiveness to touch was manifest on the left side of the abdomen. Deep tenderness was elicited about and also rather above McBurney's point. Rectal examination revealed acute tenderness well within the reach of the finger tip and toward the right side. Vaginal examination confirmed the finding. The pelvic organs appeared to be normal. The temperature was 37·4° C. (99·4° F.). There was no marked disturbance of the pulse rate.

A tentative diagnosis of subacute appendicitis was made and the patient was advised to rest and to go to bed upon a recurrence of the symptoms. A sister nurse at the institution was asked to keep her under observation. The patient had been habitually constipated and as there had been a history of thread worms within recent months, she was advised to take a course of liquid paraffin and a vermifuge.

On November 12 the patient was again seen by the writer some hours after the onset of an attack. The same physical signs as observed previously were again in evidence, the rectal tenderness and the muscular guarding were more pronounced, together with a diffuse upper abdominal soreness, and the tenderness at McBurney's point was less noticeable. There was no reference of pain to the umbilical region, nor had there been at any time. The patient was placed on board a schooner next day and taken to Kieta, one hundred miles distant, as there is not a hospital for Europeans at Buka Passage. On the morning of November 14 she was admitted to the Government Hospital for Europeans at Kieta. In consultation with the Government Medical Officer at this institution (Dr. C. M. Deland), it was decided to institute expectant medical treatment. Water and glucose only were given by mouth for five days, and the patient remained in the Fowler position. Several small doses of morphine were given on the first day and then discontinued. The patient remained in the hospital seven days, the temperature reaching 37·6° C. (99·8° F.) on one occasion and the pulse rate being above 80 on one occasion only. A somewhat puzzling feature of the case at this stage was the low pulse rate, which at the time of admission to hospital was 65. Following the rest in bed the abdominal tenderness moderated and the pain was felt only intermittently and the constitutional condition improved. The patient was a passenger by the motor vessel *Malaita* from Kieta for Rabaul on November 21. On that evening the pain was severe.

The patient was admitted to the European Public Hospital at Rabaul on November 23. She was there seen in consultation by Dr. Brem, of the Sacred Heart Mission, and by Dr. E. T. Brennan, Chief Medical Officer of the Territory. White blood counts, carried out on November 24, 26 and 28 by Dr. Backhouse, Government Pathologist, gave values of 8,240, 10,360 and 7,760, negativing the hypothesis of an abscess, which had been raised by the extreme degree of tenderness on rectal examination, coupled with a sense of fluctuation. An X ray examination, carried out by Dr. H. Champion Hosking, Government Medical Officer, indicated the presence of an appendix in the retrocaecal position, a supposition which was confirmed by the finding at operation on December 1. The operation was performed by Dr. Brennan, assisted by Dr. Hosking, Dr. Price, of the Government medical staff, acting as anæsthetist. An approach by a McBurney incision revealed, after some search, an appendix lying retrocaecally, but lacking in its proximal part a distinct mesoappendix, the organ lifting up peritoneal veils on either side near the butt. The organ traced down followed an abnormally long course and appeared to be in relation more distally with the small bowel and to have its tip at a distance of about 15·0 centimetres (six inches), situated at the brim of the pelvis. Consequently it was severed close to the butt and dealt with according to the usual technique and dissected out. Toward the distal end a mesoappendix was present. There were no signs of pus formation or of adhesions, and exploration of the pelvis showed the pelvic organs to be healthy. The tip of the appendix appeared to be somewhat firmer than the remainder of the organ and yellowish in hue. Otherwise, as far as could be judged at the time, it did not appear unhealthy, but, unfortunately, the specimen, owing to an error on the part of an attendant, was not preserved for pathological examination. The patient made an uninterrupted recovery and left the hospital twenty-three days after admission.

The case is cited:

1. To illustrate the value of leucocyte counts in excluding the presence of an abscess.
2. To show the value of X rays in indicating the position in which an operator may expect to find the appendix.
3. Because of the unusual length of the appendix in an adult.
4. Because of the disproportion between the pain and the condition of the appendix seen at operation.

Reviews.

EXPERIMENTAL PHYSIOLOGY.

"EXPERIMENTAL PHYSIOLOGY FOR MEDICAL STUDENTS", by Dr. D. T. Harris,[1] is a revised and enlarged edition of "Practical Physiology", by G. V. Anrep and D. T. Harris, which appeared in 1923. Changes have been made in many of the experiments, and a number of new experiments have been added.

The range of observations covered is very large, and any medical student who worked conscientiously through all of them would have a very sound knowledge of experi-

[1] "Experimental Physiology for Medical Students" (being the revised and enlarged edition of Anrep and Harris' Practical Physiology), by D. T. Harris, M.D., D.Sc., F.Inst.P.; Second Edition; 1934. London: J. and A. Churchill. Royal 8vo., pp. 248, with illustrations. Price: 12s. 6d. net.

XUM

mental physiology. It would, however, not be possible for any student to do all the experiments set out in the time usually allotted for this work. Many useful demonstrations are also described.

The descriptions of experiments are frequently too brief and would require to be amplified by the demonstrator if the student was not to waste a considerable amount of time.

Some of the experiments, such as spectroscopic examination of blood pigments, analysis of gastric contents, air analysis, experiments in tissue respiration, would be better treated in a course of biological chemistry.

There are also ten pages of simple exercises in pharmacology which cover an excellent range.

While one may criticize the book in details, there is probably no better manual of experimental physiology at present available in English.

THE HORMONES.

"THE CHEMISTRY OF THE HORMONES", by Benjamin Harrow and Carl P. Sherwin, deals for the greater part with the chemistry of the products of the endocrine glands, including methods of preparation, chemical properties and, where known, chemical constitution of the active principles.[1]

The physiological effects of these principles and their clinical applications are treated very briefly. The hormones dealt with are the thyreoid hormone, the parathyreoid hormone, insulin, the pituitary hormones, the adrenal hormones, the male hormone, the female hormones, secretin and plant hormones. The account of each hormone is prefaced by an excellent summary of the history of the discovery and separation of the particular hormone. Very full details are given of the different methods which have been used in separating the several hormones and in their standardization.

Each section is followed by a good and representative bibliography. Such physiological data as are included have been selected with care without the overstatement which is so usual in books on endocrinology.

The book is essentially a laboratory manual and will be very useful to laboratory workers who wish to prepare solutions of the hormones and to study their effects, but it will be of little use to the general medical reader.

PROGRESS IN ENDOCRINOLOGY.

PROFESSOR A. T. CAMERON, who occupies the Chair of Biochemistry at the University of Manitoba, has produced, in his "Recent Advances in Endocrinology", an important contribution to the valuable "Recent Advances" series published by J. and A. Churchill.[2]

In this volume all the important recent biochemical and experimental work in connexion with the endocrine glands is fully described. In the preface the author states: "Endocrinology is essentially a biochemical subject to this extent: The precise truth of its teachings depends ultimately upon the isolation of the different endocrine principles in pure crystalline form so that their physiological and pharmacological properties may be ascertained accurately"; and although he apologizes that he, a biochemist, should stress his own ideas in dealing with the clinical aspect of the subject, he regards this aspect as being of perhaps the most importance.

The author gives an account of the biochemistry, physiology and pathology of the various glands, and describes the clinical features associated with their disorders. In successive chapters the thyreoid gland and iodine metabolism, the parathyreoid glands, the islets of Langerhans and insulin, the adrenal glands, the pituitary gland and the endocrine secretions of the organs of reproduction are dealt with. Reference is made to some actual and presumptive endocrine principles in the penultimate chapter, in which the classical work of Bayliss and Starling, and J. Mellanby's more recent experiments in connexion with secretin are described, and Laughton and Macallum's observations with regard to a possible principle stimulating the production of insulin are discussed.

The much debated question of endocrine interrelationships is left to the last chapter. The rarity of true pluriglandular disorders is rightly stressed. At the conclusion of each chapter a large number of references is appended.

The scientific aspect of endocrinology is always kept to the fore, and the dangers of loose thinking are indicated. In the latter connexion the author quotes Cushing as follows: "Endocrinology lends itself to two glaring faults, one the popularization of writing on the subject, and the other a tendency of clinical observers to draw upon their fancy in a symptomatology which does not lend itself to precision."

Unfortunately treatment is not stressed in this volume, but the author reminds his readers that rational replacement therapy must always take into account that only two or three endocrine principles have been definitely demonstrated to be effective when administered by mouth.

Every practitioner who wishes to learn the truth about the various endocrine principles, of which he hears so much from the commercial travellers who haunt his rooms and who exhort him to prescribe an extract of this organ or that organ in order to produce a miraculous change in his patient, should study "Recent Advances in Endocrinology".

[1] "The Chemistry of the Hormones", by B. Harrow, Ph.D., and C. P. Sherwin, D.Sc., M.D., D.P.H.; 1934. London: Baillière, Tindall and Cox. Royal 8vo., pp. 234. Price: 11s. 6d. net.

[2] "Recent Advances in Endocrinology", by A. T. Cameron, M.A., D.Sc., F.I.C., F.R.S.C.; 1933. London: J. and A. Churchill; Australia: Angus and Robertson. Demy 8vo., pp. 373, with illustrations. Price: 22s. 6d. net.

Notes on Books, Current Journals and New Appliances.

MATERIA MEDICA FOR NURSES.

OBVIOUSLY a nurse should know something of the action of the drugs that her patient is taking. Whether she should know much or little is a matter of opinion. Two books have recently been published with the object of supplying information to nurses.

The first is quite a comprehensive work. It is written by Miss Lois Oakes, a nurse, and Mr. Arnold Bennett, a pharmacist.[1] It has been written with the object of covering the syllabus of the General Nursing Council and "to enable the student nurse to gain an adequate knowledge of the subject". It covers practically the whole of the subject, certainly as much, if not more, than any nurse should be expected to know. Some of the chapters are likely to be very useful, notably that on the prescription, in which symbols and signs are explained, that on posology, and the chapter on synonyms of common drugs. In the chapter on common drugs and preparations the drugs are described, their action is stated, and preparations containing them are enumerated. This chapter would be specially useful to any nurse as a chapter of reference.

The second book is less pretentious; it is intended not only for those preparing for examination, but also "as a handy book of reference after qualification".[2] Only those drugs in common daily use are dealt with; their important preparations are stated, with their doses, actions and uses. The author, Dr. A. Muir Crawford, of Glasgow, has made no attempt to present a complete list of drugs, but he has made a wise and careful choice.

Both these books may be recommended to student nurses and to those who have gained their certificates.

[1] "Materia Medica for Nurses", by L. Oakes, S.R.N., D.N., and A. Bennett, M.P.S.; 1934. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 347. Price: 7s. 6d. net.

[2] "Materia Medica for Nurses", by A. M. Crawford, M.D., F.R.F.P.S.G.; Third Edition; 1934. London: H. K. Lewis. Crown 8vo., pp. 110. Price: 3s. 6d. net.

The Medical Journal of Australia

SATURDAY, MARCH 16, 1935.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE LEAGUE OF RED CROSS SOCIETIES.

AN account of the seventeenth International Red Cross Conference, held at Tokyo, Japan, in 1934, has been published in the December, 1934, issue of the *League of Red Cross Societies Monthly Bulletin*. The League of Red Cross Societies is an organization that should wield a great influence on the outlook of nations. It was founded more than fifteen years ago by Mr. Henry P. Davison, who began by calling together several Red Cross societies of different countries. Since then it has grown, and now it comprises sixty-one national societies. When the league was founded in May, 1919, Mr. Davison expressed the belief that the manifestation that would be made through the league would result in the establishment of closer relationship between nations, a better understanding and therefore greater confidence, which would in turn result in better conditions and in increased happiness and contentment throughout the world. The project was ambitious, but the objective was worth striving for. The organization should therefore be widely known, appreciated and helped; particularly should medical practitioners take cognizance of it, for many of its activities are directed towards the protection of health.

During the Great War every combatant country had its Red Cross society or some similar body. The organization of these bodies showed that people of all types could combine to give service to others. The stimulus of war was the driving force; every nation believed its cause was just and the individual in the community believed what the leaders in the country wanted him to believe. That was war, and war will probably always be the same. That the war-time efforts should be turned into peaceful channels and that erstwhile combatants and others should be brought into friendly union, with common objectives, was a magnificent idea. Some of the activities reported at the recent conference show that concerted action has been taken. An International Relief Union has been formed and steps have been taken to organize relief in cases of disaster. Red Cross societies have been urged to improve the relief organizations in their own countries, to respond to appeals from international Red Cross bodies, and to keep the Secretariat of the League of Red Cross Societies fully acquainted with the development of their work, so that they can help the Secretariat to discharge its responsibilities.

Much has been done in the sphere of health. The health of seamen has always been the concern of the Red Cross bodies. On previous occasions we have described this work. A manual dealing with health has been published in the French language; it is proposed to have this manual translated into other languages and to have it adopted in different countries. Efforts are being made to have medicine chests for use at sea standardized, and also to urge the standardization of medicine chests for the air services. Air ambulances have been discussed and the introduction of regulations and technical facilities has been urged to enable ambulance aeroplanes to cross frontiers in times of peace. The League has stimulated action in many countries to combat tuberculosis and venereal disease; and in many places the Red Cross societies have taken a prominent part in the fight against disease, for example: cancer in Germany; malaria in Italy, Armenia, Greece, Holland and the Congo; trachoma in Russia and Poland; pellagra in America; leprosy in the Congo and the Gold Coast. In addition, much

of the discussion at the recent conference was concerned with the civilian in time of war. The League is obviously concerned with efficiency of medical services in case of war, but at the same time it urges each of its component parts to "exert every effort, within the sphere of its attributions, to prevent war". This will no doubt be described as a pious resolution, for in the account of the conference as published no ways or means of attaining this object are suggested.

The League of Red Cross Societies is thus seen to cover a wide range of interests. In Australia Red Cross organizations exist in every State. According to the report of the Secretary-General of the League, the Junior Red Cross in the Commonwealth has a membership of 90,000 school children, and these children correspond with children in twenty-two other countries. It is in the juniors that most reliance must be placed. If suffering humanity is to be served, if greed of men and the lust for power are to be curbed, it is with the children of the community that the start must be made. One of the leaders of the League of Red Cross Societies has described it as a child of post-war idealism; it is one of the means that may be used to instruct the young.

Current Comment.

CARDIAC ŒDEMA.

Many theories of the causation of cardiac œdema have been evolved and elaborated; those not yet discarded are still little more than theories. Considerable attention has been given lately to the possible influence of the plasma protein content on the exchange of fluid between the capillaries and the tissues. Observance of a lowered plasma protein content in diseases accompanied by œdema is not new; as long ago as 1836 Richard Bright noted that the blood plasma of persons suffering from Bright's disease was deficient in albumin. Within the past few years similar observations have been made in œdema of cardiac origin. William A. R. Thomson has recently published the results of his investigations of the blood plasma content in a series of 54 patients—18 with heart failure and œdema, 16 with heart failure and no œdema, six with Bright's disease, four with tuberculosis, seven with other diseases, and three normal persons.[1] His figures for normal persons approximate those obtained by other observers. He calculated the colloid osmotic pressure from the figures supplied by Govaerts, and this, too, he found to vary but little from what other workers have regarded as normal.

In the group of patients suffering from cardiac failure and œdema, he found that the plasma protein content was as a rule considerably below normal. This deficiency was mainly in the albumin content; the globulin content remained about normal, and in some cases was actually above normal. The colloid osmotic pressure tended to be low. Thomson points out that the colloid osmotic pressure may still be low when the plasma protein content is higher than normal, providing the albumin content is low. He notes that 87·5% of the patients with cardiac œdema had a plasma protein content of less than 3·2 grammes per 100 cubic centimetres, and 91% of them had a colloid osmotic pressure of less than 29 centimetres of water. In the group of patients suffering from cardiac failure without œdema, the plasma albumin content tended to be lower than normal; but it was above 3·2 grammes per 100 cubic centimetres in 87% of cases. In the same group the colloid osmotic pressure was above 29 centimetres of water in 91% of cases. Thomson arbitrarily calls these (albumin content of 3·2 grammes per 100 cubic centimetres and colloid osmotic pressure of 29 centimetres of water) the "critical levels" for albumin content and colloid osmotic pressure respectively. It is striking that in the group with œdema the albumin content was below the critical level in 87% and in the group without œdema it was above the critical level in 87% of cases; similarly, in the first group the colloid osmotic pressure was below the critical level in 91%, and in the second it was above in 91% of cases. In the miscellaneous group without œdema the albumin content and the colloid osmotic pressure were always above the critical levels. In the group with pulmonary tuberculosis the figures were also low, and it was noted that in the three cases with effusion or œdema they were considerably below the critical levels. In the group with nephritis the figures were below the critical levels in each case in which there was œdema. The results of the investigations in this group agreed with those obtained by other workers.

Thomson remarks that the "importance of the plasma proteins lies in their relation to the capillary pressure". The hydrostatic pressure in the capillaries tends to drive fluid into the tissues, and the colloid osmotic pressure (or "onkotic" pressure) tends to draw fluid from the tissues into the capillaries. Normally a nice balance exists between these two forces. If the hydrostatic pressure is greatly raised or if the onkotic pressure is greatly lowered, œdema may occur. He points out that in nephrotic œdema there is a pronounced fall in the onkotic pressure, the hydrostatic pressure remaining comparatively unaffected; in cardiac œdema there is an increase in the hydrostatic pressure as well as

[1] *The Quarterly Journal of Medicine*, October, 1934.

a moderate fall in the onkotic pressure. In either case there is a disturbance of the balance. This theory explains why œdema may occur when there is only a slight diminution in the plasma protein content. Thomson goes on to say: "The other factor which undoubtedly plays a part is that of capillary permeability which, it is practically certain, is increased in these cases." He expresses the opinion that malnutrition is the cause of plasma protein deficiency in cardiac œdema. He states that this malnutrition is due partly to the disease itself, partly to insufficient protein intake and partly to interference with protein formation. It has been suggested that the apparent fall in the plasma protein content is due to increase in blood volume; but Thomson shows that this is not true, by pointing out that it is the albumin only that diminishes in quantity, while the globulin content remains about normal or is actually above normal. He points out also that albuminuria could not account for the depletion of plasma albumin in his cases, as it was never sufficiently pronounced to have any appreciable effect. He concludes with the suggestion that persons with cardiac œdema should be given a diet rich in protein, restriction of salt and fluid intake being observed as at present.

This is an important piece of work, if only because it confirms in Great Britain the observations made by investigators in other parts of the world. It does more than this however, as it draws closer attention to the possible influence of the plasma proteins in the prevention of œdema, and provides strong evidence in support of the theory that œdema is partly or mainly due to a relative increase in the hydrostatic pressure in the capillaries or a relative decrease in the colloid osmotic pressure. This is the most attractive theory that has yet been advanced.

CANCER OF THE BRONCHUS AND LUNG.

The December, 1934, issue of *The Journal of Thoracic Surgery* is devoted to a series of papers on cancer of the bronchus and lung, read at the annual meeting of the American Association for Thoracic Surgery in 1934. The first paper is by Tudor Edwards, of London. It is based on 73 patients treated by radical operation or by radon seeds introduced either through the bronchoscope or at thoracotomy. There was the usual preponderance of males (53 of the 73), and 62 of the 73 patients were between forty and seventy years of age. The commonest site was the lower lobe, both sides being almost equally affected. Edwards discusses the symptoms and signs and correlates these with the findings on X ray, bronchoscopic and thoracoscopic examination. The diagnosis is sometimes difficult; for example, in a series of patients operated on for chronic pulmonary abscess there was an underlying carcinoma in 10%. Edwards has treated 32 patients by radon seeds placed near the growth in a special container introduced through the bronchoscope. Most of these people have died of the disease, but he has had some encouraging results; for example, one patient is alive and well three years afterwards, and several others for lesser periods. Twenty-seven patients had radon seeds put into the growth after it had been exposed at thoracotomy, radical operation having been found impossible. Here also some of the results justified the procedure; for example, one patient is alive and well four years later, one two years later, and one twenty months later. Fourteen patients had radical operation; in two the whole lung and in the rest a lobe only was removed. Five are still alive, the interval since operation varying from six years to six months. The remaining nine died either from shock, hæmorrhage or sepsis soon after operation, or later from metastases. Considering the nature of the disease and the operation, the results must be regarded as encouraging, and it is not too much to hope that more patients in the future will be given the chance of cure offered by operation.

Further support for some such active treatment is found in two other papers in the same journal. Richard Overholt reports successful total pneumonectomy in two female patients for proved carcinoma. Both were symptomless and apparently free from metastases one year and six months respectively after operation. Carl Eggers records an immediately successful lobectomy for carcinoma in a woman aged forty-five; but here the ultimate prognosis is bad, because there were nodules of growth on the parietal pleura; these were irradiated after the patient had recovered from the operation, but it was too early to know the final result. In the subsequent discussion E. D. Churchill said that he had removed the whole lung from three patients for cancer. One died on the tenth day from perforation of multiple acute ulcers of the duodenum, and one at a later date from metastases; the third patient is apparently cured. Other successful cases are on record; in a previous issue we commented on one of the first total pneumonectomies reported, the patient being a medical practitioner operated on by E. A. Graham and J. J. Singer.

Chevalier Jackson, junior, and F. Konzelmann describe the bronchoscopic appearances in 32 cases and give coloured illustrations of some of them. They describe the technique of securing material for biopsy. Their pathological classification is as follows: (i) adenocarcinoma, (ii) squamous cell carcinoma, (iii) undetermined type of carcinoma, (iv) indifferent basal cell carcinoma. By bronchoscopic examination one can also define the level of the growth and form an idea as to its operability and to the type of operation possible.

There are two papers on the classification of lung tumours in relation to treatment and prognosis, but the opinions in each paper are in many points so divergent that it is clear that further work on this aspect of lung growths is necessary before definite conclusions can be made.

XUM

Abstracts from Current Medical Literature.

PÆDIATRICS.

Sedimentation Rate in Rheumatic Carditis.

C. BRUCE PERRY (*Archives of Disease in Childhood*, October, 1934) has carried out 1,043 sedimentation rate estimations on 167 children suffering from rheumatic heart disease. The children were divided into five groups, namely: those in whom there was no clinical evidence of active rheumatic infection at the time of the examination; those who showed chorea as the only sign of rheumatism, and with no carditis; those who had chorea with carditis; those with monocyclic types of attacks or relapses of carditis without chorea as an initial feature; and those with polycyclic types of attacks or relapses. In the first group over 85% were under 10 millimetres in the first hour. In the second group, with the exception of two patients, the sedimentation rate was normal in all instances. The author draws attention to this observation in view of the fact that chorea is generally regarded as an active rheumatic manifestation. In marked contrast, the majority of patients suffering from chorea with carditis had a high sedimentation rate. In both groups in which the rates were determined during attacks or relapses of carditis, the reading was never below 15. In two of these patients congestive cardiac failure developed while they were under observation, and the sedimentation rate became normal, although the disease was obviously active; in one who recovered the rate was raised again as the œdema cleared. The author explains this anomaly by a change in the plasma proteins coincident with the œdema, in view of the evidence that these play an important part in regulating the sedimentation rate of the red blood cells. The author concludes that acute rheumatic carditis is always associated with a high sedimentation rate, and that, although a high rate in a child with rheumatic heart disease does not necessarily indicate active infection, it should lead to a careful investigation to exclude such activity.

Bone Changes in Leuchæmia.

CHARLES E. SNELLING AND ALAN BROWN (*Archives of Disease in Childhood*, October, 1934) report a series of twelve cases of lymphatic leuchæmia with changes in the structure of the long bones occurring in children under six years of age. The changes referred to are a rarefaction across the shaft just adjacent to the epiphyseal line, occurring in eight of the series, and periosteal elevation, occurring in four. The children of the latter group had pains in the extremities and the symptoms simulated rheumatism or scurvy. Osteomyelitis has also to be considered in the differential diagnosis. Further, the periosteal changes might be confused with the picture of luetic bone disease. The authors hold that if a child has a blood picture suggestive of leuchæmia, a radiological examination of the long bones is of value in confirming the diagnosis; and that, in conditions in which symptoms of rheumatic fever are present, an examination of the blood and long bones may be necessary to exclude leuchæmia.

H. Erb, in an accompanying paper, describes the pathological changes occurring in bone in leuchæmia. Just as infiltration of the liver and kidney or other tissues with leuchæmic cells may occur, so may the marrow cavity, Haversian canals, or subperiosteal region in bones be involved. A profound anæmia may result from replacement of the bone marrow by these cells. Infiltration is not visible on X ray examination. Rarefaction occurs chiefly towards the end of the long bones and may cause spontaneous fractures. As a result of the infiltration of the subperiosteal region, proliferation of new bone occurs beneath the periosteum. This new bone and the rarefied areas are demonstrable by X ray examination.

Repeated Sedimentation Tests.

LEE D. VAN ANTWERP (*American Journal of Diseases of Children*, October, 1934) records his observations on repeated erythrocyte sedimentation rate estimations carried out as a routine measure on all new patients admitted to a children's sanatorium for tuberculosis. He points out that sedimentation rates in children tend to vary widely without intervening clinical causes, and that erroneous interpretations may result accordingly. He considers that apprehension on the part of the patient may be a factor in these variations and that when a high rate is first recorded it may be necessary to repeat the estimation, just as it is necessary at times to repeat metabolic tests. The test carried out at the time of the greater excitement gave the higher reading. The author discusses the possibility of the influence which increased activity of the suprarenals, due to excitement or apprehension, may have on the sedimentation rate. He added epinephrine to a specimen of blood and estimated the sedimentation rate, while the sedimentation rate of a second specimen drawn from the same patient at the same time was estimated in the usual manner as a control. There was no great variation in the two rates. The possibility of increased glycogenolysis with raised blood sugar, acting through the medium of excessive secretion of epinephrine resultant upon excitement or apprehension, was also considered. A solution of dextrose was added to blood in the process of sedimentation, and the reading was compared with a control, but this attempt to check the possible influence of the suprarenals was likewise not productive of results. The author points out, however, that the dextrose of the blood is probably not identical with the dextrose known in chemistry, and that the observation is accordingly not conclusive. Further observations are being made to check the sedimentation rates with blood sugar readings of specimens of blood drawn at the same time, and they give promise of showing a fairly consistent increased blood sugar content with increased sedimentation rates.

Tumours of the Brain in Infancy.

SIDNEY W. GROSS (*American Journal of Diseases of Children*, October, 1934) has investigated the symptomatology, mode of development and histological nature of primary neoplasms in the brain or its coverings in infancy. The study is based on nine examples of such tumours occurring in children in the first and second years of life. Eight of the nine patients were males, and this is in keeping with other reports in the literature of tumours of the brain in infancy, which record a predominance of the male sex. Hydrocephalus was present in every instance, vomiting in all but two, choked disk in five, and engorged retinal veins in three. Seven of the tumours were in the mid-cerebellar region, this site having been previously mentioned by Cushing as being the one of election for tumours of the brain in early life. Three of the tumours were ependymomata, one was a pineoblastoma, one was an astroblastoma, one was an astrocytoma, two were medulloblastomata, and one was an embryonic tumour of neuro-epithelial origin which could not be classified further. The author draws attention to the difficulty in the diagnosis of these tumours owing to the age of the patient precluding the testing of visual fields and sensation and the use of other tests which require the cooperation of the patient. It is therefore necessary to depend more on the history and on observations of the general behaviour of the infant. The symptoms in infancy frequently develop with amazing rapidity, particularly the sudden and rapid enlargement of a previously normal head. The problem of diagnosis largely resolves itself into a differentiation of hydrocephalus due to tumour from one due to other causes, notably obstruction of the aqueduct as the result of either an inflammatory process or a congenital defect. Subdural hæmatomata often cause enlargement of the head and are more frequent in infants than has been realized in the past, but by careful attention to the history and clinical studies this condition can usually be differentiated from tumours. Abscess of the brain, though it occurs, is rare in infancy, and a history of a discharging area or a septic process else-

where in the body should suggest such a possibility. The author considers that ventriculography is not so helpful in infants as in adults.

ORTHOPÆDIC SURGERY.

Coxa Plana and Related Conditions at the Hip.

A. B. FERGUSON AND M. B. HOWORTH (*The Journal of Bone and Joint Surgery*, October, 1934) include in their study of *coxa plana* slipping of the upper femoral epiphysis, *osteochondritis dissecans*, a certain group of cases of chronic degenerative arthritis, and a condition not previously identified, as a clinical entity to which the name *coxa magna* is given. They are not primary diseases, but are the effect of an underlying ischæmia, the cause and distribution of which may vary. The condition resulting from the ischæmia is determined by the age of the patient, the distribution and degree of ischæmia and, to a slight extent, by sex and some minor factors. In their cases of *coxa plana* and related conditions ischæmia has been found to arise from several causes, classified and described as: Type 1, inflammation in the hip joint; Type 2, inflammation or, rarely, tumour adjacent to the joint, usually in the femoral neck; Type 3, fracture or epiphyseal separation at the femoral neck; Type 4, trauma to the femoral head; Type 5, tension of the soft tissues about the femoral neck. They consider that ischæmia at the femoral head may result from any one of the several causes described and may cause one of the conditions related to *coxa plana* in one patient, a different condition in another patient, and no sequela at all in a third patient, depending chiefly upon the age and sex of the patient and the degree of ischæmia. In the first decade of life the substance of the femoral head is more susceptible to the influence of a circulatory disturbance; in adolescence the growth disk is more likely to be affected. In an age period at which the growth disk is vulnerable to the presence of a moderate degree of ischæmia, the substance of the head may be vulnerable to a greater degree of ischæmia, whereas neither may be vulnerable to a lesser degree of ischæmia. Vulnerability of a given part of the hip occurs some two or three years earlier in life in girls than in boys. The relationship between vulnerability of the part and the type, degree and distribution of the ischæmia determines whether *coxa plana* or a different but related condition shall develop. They consider that subacute arthritis may occur and subside without a sequela in cases where the degree of involvement is slight or the hip is not vulnerable. In such cases there are, for a short time, symptoms and signs which suggest the onset of *coxa plana*, but these clear up without further changes. The cases are classed as abortive if *coxa plana* or reslipping epiphysis actually develops, but resolves after slight changes occur. Broad, shallow and sometimes irregular femoral heads occur in some forms of chondrodystrophy. These are not to be confused with *coxa plana*, which is characterized chiefly by irregular ossification of the head and by symptoms and signs of inflammation which are not present in chondrodystrophy unless complicated by actual *coxa plana* or a related condition.

Posterior Displacement of Fifth Lumbar Vertebra.

A. D. SMITH (*The Journal of Bone and Joint Surgery*, October, 1934) states that posterior displacement of the fifth lumbar vertebra on the sacrum usually causes severe pain, is disabling to a considerable degree, and is but little affected by the usual methods of conservative treatment. His study is based upon fifty-six patients. The average age at which they presented themselves for treatment was thirty-one and six-tenths years, but the average age when symptoms first began was twenty-nine years. This corresponds quite closely to the age at which symptoms begin in other abnormal mechanical conditions in the lumbo-sacral region. The author considers that unquestionably the most important single factor in determining a posterior displacement is the structure of the lateral articulations between the fifth lumbar and first sacral vertebræ. Trauma plays but a secondary rôle and can cause such a displacement only if a certain mechanical set-up is present. The posterior displacement is not necessarily great. Its presence in any degree is indicative of a very unstable joint and of the possibility of a temporary increase in the displacement from an unusual strain. It seems quite probable that an actual sprain of the ligaments of these joints also could be easily produced. In operations on these patients it regularly was found that the mobility of the fifth lumbar vertebra on the sacrum was much greater than normal. An important secondary factor in causing posterior displacement is diminution in thickness of the intervertebral disk, probably through rupture of the *nucleus pulposus*. Posterior displacement was not present, however, in all cases of thinning of the disk, and he considers that oblique or antero-posterior articulations probably are a necessary concomitant. Radiation of the pain along part or the whole of the sciatic distribution was present in thirty patients. This was a striking and characteristic feature of the symptomatology in these cases, and the explanation lies in the concept of pressure upon the fifth lumbar root caused by the pedicles and the lateral articulation. A fairly definite clinical picture was found in many cases of posterior displacement of the fifth lumbar vertebra, including a history of low back pain with radiation to the back of the thigh and outer side of the leg, recurring in acute attacks; tenderness over the lumbo-sacral region; muscle spasm and limitation of motion, and often a lateral list of the trunk. The decisive and most important factor in the diagnosis, however, is the radiograph. The author sums up his study with the statement that conservative measures seldom cure the symptoms from posterior displacement of the fifth lumbar vertebra and, in the majority of cases, lumbo-sacral fusion is the only effective means of treatment.

Slipped Upper Femoral Epiphysis.

W. E. BROGDEN (*The Journal of Bone and Joint Surgery*, January, 1935), in his series of fifty-seven cases of slipped upper femoral epiphysis, found a larger percentage to be in the thin, tall type of individual with no evidence of disease or disturbance of growth; and in the majority of case there was a definite history of trauma with immediate onset of disability. Whether or not a good open or closed reduction had been attained, the post-operative skiagrams seemed to bear no relation to the functional end results. With these points in view, all available cases of slipped upper femoral epiphysis from 1928 to 1933 were examined, reviewed and the skiagrams were re-read. End results were designated excellent when there was present no limping, pain, stiffness or limitation of motion. In 27 of the 57 cases trauma was the sole causative factor. Abnormality of growth and endocrine imbalance, either separately or in conjunction, were designated as predisposing ætiological factors in 34 of the 57 patients. Nineteen cases in which there were no signs of predisposing pathological change were classed as cases of incomplete epiphyseal fracture of the neck of the femur, the exciting cause being direct or indirect trauma. This type of injury, if treated in the same manner as fractures in adults, namely, by closed reduction, does not tend to result in increasing deformity. This deformity is dependent upon the activity of the patient, weight-bearing, and the physiological weakness of the epiphyseal line. Closed manipulation by the Whitman method in any case in which there was a history of less than four months' duration, gave the best result. Twenty of the patients in the series had good end results. Traumatic arthritis was seen to occur as frequently in the patients subjected to open operation as in those subjected to repeated stretchings and manipulations. The author considers that all patients are entitled to the benefit of the doubt and that an attempt at closed reduction should be made if disability is of less than four months' duration. If this is unsuccessful, an open operation should be performed. In all cases of over four months' disability the patients are entitled to an open operation as a primary procedure, except in selected cases.

XUM

British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held at Saint Vincent's Hospital, Melbourne, on November 21, 1934. The meeting took the form of a series of demonstrations by members of the honorary staff.

Intracapsular Fracture of the Neck of the Femur.

Dr. Thomas King discussed the treatment of intracapsular fracture of the neck of the femur. He said that the choice of adopting either the non-operative method of Whitman or one of the various operative measures was by no means settled, but that present surgical opinion tended to favour the method of fixation by the Smith-Petersen nail. The greater problem was whether the nail should be introduced after arthrotomy and direct visualization of the fracture or by closed reduction and nailing the fragments by a small incision over the junction of the shaft with the base of the trochanteric region. Dr. King said that while he was keeping an open mind on this question and was prepared to admit that arthrotomy had a place in the treatment, he was using a subcutaneous method of directing the nail. He used a Smith-Petersen nail canaliculized by a hole, 2·3 millimetres ($\frac{3}{32}$ inch) in diameter, along the whole length of the nail, at the junction of the three blades or fins. A Kirschner wire introduced and checked by X ray control acted as a guide along which the nail could be accurately driven into the head. Having used this method eighteen months previously for the first time, he had subsequently learnt that Johansson, Nicklas, Jerusalem (who used a nail with a hole in the head only) and others were also using a Kirschner wire drilled into the neck and head of the femur as a guide for the nail. Dr. George Clemons, of Launceston, had also suggested this method.

Whilst many were following Johansson's technique for obtaining the surface anatomy of the head and neck of the femur, others were working on different lines. That the markings were not entirely satisfactory was borne out by the multiplicity of methods for determining them. Dr. King did not think that the neck of the femur could always be made to lie horizontally to the operating table by internal rotation of the foot for 45°. Dr. King said that his treatment was different from all others in that the wire was placed behind the neck, under fluoroscopic control, and formed the basis for guidance of the actual wire in the bone itself. He now drilled two wires into the bone and chose the better one as a guide, as shown by skiagrams in antero-posterior and lateral views of the hip.

A cinematograph film of the complete technique was shown. It was seen that the nail was introduced along the guide wire in the bone with the "V" of the blades looking cranially, so that the broadest surface of the nail lay on the dense *calcar femoralis*. Dr. King said that the ideal place for the nail was in the lower one-third of the neck. After four weeks' traction on a Braun splint the patient could go home and lie about on a sofa or sit on a chair. Weight-bearing before the lapse of eight to twelve weeks was forbidden. The nail became loose after six months and should then be removed.

Tetany Following Thyreoidectomy.

Dr. J. W. Grieve showed a girl, aged seventeen years, who had been admitted to hospital on January 6, 1933, with thyreotoxic symptoms of three months' duration: nervousness, restlessness, palpitation, shortness of breath, excessive appetite and amenorrhœa for two months. There had been no loss of weight. Swelling of the neck had been present for three months. There was a previous history of recurrent sore throats. She had had appendicitis seven years before. The family history revealed that one sister had had swelling of the thyreoid gland.

Examination on admission had revealed diffuse enlargement of the thyreoid gland. The heart was enlarged, the apex beat being in the fifth intercostal space, 11·25 centimetres (four and a half inches) from the mid-sternal line. The heart sounds were normal. The pulse rate was 120 per minute and the blood pressure 140 millimetres of mercury systolic and 70 diastolic. The basal metabolic rate was +28. A fine tremor was observed. There were slight prominence of the eyes and some lagging of the lids; convergence was poor. The skin was moist and the tonsils were enlarged.

On January 25, 1933, she had an attack of follicular tonsillitis.

On February 13, 1933, the right lobe of the thyreoid gland was removed under local anæsthesia, the usual pre-operative and post-operative treatment being carried out. Examination of a section of the gland revealed mostly colloid acini. There were a few areas of small, newly formed acini; the fibrous stroma was increased, and there were a few foci of round cells.

On May 28, 1933, the patient was readmitted to hospital because she was still nervous and irritable, her heart action was rapid, and her thyreoid gland was increasing in size. Examination at that time showed that the left side of the thyreoid gland was rather prominent. Her pulse rate was 106 to 156 per minute and her systolic blood pressure 160 millimetres of mercury. Tremor was again observed.

On June 15, 1933, the left lobe of the thyreoid gland was removed. The usual pre-operative and post-operative treatment was carried out. The pulse rate fell to 90 or 100 per minute; but the blood pressure remained at 160 millimetres of mercury. The patient was discharged from hospital after fourteen days.

On January 23, 1934, she was readmitted. Her thyreoid gland had been increasing in size for the previous four months. She was irritable and restless. There was a history of sweats, but no loss of weight. Her menstrual periods were irregular. Examination at that time showed that the eyes were still rather prominent, and there was slight lagging of the lids. The thyreoid gland was uniformly enlarged. The apex beat of the heart was in the fifth intercostal space, ten centimetres (four inches) from the mid-sternal line. The first sound was a slapping one. The pulse rate was 100 to 120 per minute and the blood pressure 140 millimetres of mercury. Tremor was observed.

On January 25, 1934, further thyreoidectomy was performed, accompanied by the usual pre-operative and post-operative treatment. On January 28, 1934, she complained of numbness and stiffness in the right hand. On January 29, 1934, she was drowsy, weak and restless. Tetany was pronounced. On January 30, 1934, she was given ten cubic centimetres of a 10% solution of calcium chloride intravenously, also calcium lactate in a dose of two grammes (thirty grains) three times a day, and parathyreoid extract in a dose of 0·006 gramme (one-tenth of a grain) twice a day. She improved and the calcium lactate and parathyreoid were continued. On February 28, 1934, tonsillectomy was performed.

On March 3, 1934, she was readmitted to hospital three days after tonsillectomy, with severe tetany, extreme restlessness, a twitching face, and spasms of arms and legs. She was given an intravenous injection of ten cubic centimetres of a 10% solution of calcium chloride. By that time she showed signs of subthyreoidism; for her hair was falling out in large handfuls and her facial appearance was suggestive of the disorder. The administration of calcium and parathyreoid extract was continued, and the administration of thyreoid extract was commenced. Her pulse rate was 74 to 80 and her blood pressure 126 millimetres of mercury.

She was discharged to the out-patients' department and had been attending there since. She was being given calcium and sodium lactate and "Ostelin", "Adexolin", parathyreoid extract, and thyreoid extract.

Her general condition had improved and she had been relatively well. She was still nervous and irritable. Her hair had been falling out a little. Her menses were rather irregular and she showed signs of tetany at these

times. She still had occasional muscular twitching and signs of latent tetany. Her blood calcium and phosphorus content had not been estimated.

In June, 1934, blurring of the optic disks and small retinal hæmorrhages were observed; but these had cleared up.

Dr. Grieve, to whom this patient had recently been referred, asked for an expression of opinion as to the prognosis and also as regards the value of calcium by mouth. He intended to stop the calcium medication to observe the result. It would appear that it might be helpful at the time of her menstrual periods.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Renwick Hospital for Infants, Sydney, on November 15, 1934. The meeting took the form of a series of demonstrations by members of the honorary staff.

Pink Disease.

DR. INGLIS ROBERTSON showed three patients suffering from pink disease. The first was a female child, aged thirteen months, who had been admitted to hospital on October 31, 1934. She was the second child of the family, and her birth had been normal. She had had an illness described as "gastritis" ten months previously. Her diet consisted of milk and milk foods. She had not been well for two months prior to admission and had been pale and irritable. During the week immediately before admission she had passed frequent, green, slimy stools, and had suffered from anorexia and vomiting; she had been feverish and drowsy for two days. Physical examination at that time revealed that the general condition was poor; the child was thin and dehydrated and drowsy; there were photophobia and hypotonicity of the muscles. Teeth were erupting. Occasional râles could be heard on either side of the chest. During her stay in hospital the child was very miserable; the hands and feet were slightly pink at times; the photophobia became less pronounced; the weight decreased. There had been a slight improvement.

Dr. Robertson's second patient was a boy, aged eleven months, who had been admitted on September 17, 1934. He was a second child; the first child was quite well. He had suffered from recurrent attacks of bronchitis since his birth. The birth had been normal, and the weight at birth had been 4·65 kilograms (ten pounds four ounces). The diet had consisted of breast milk for six months, and at the time of admission "Lactogen" and milk foods. Four months before admission the feet and hands had become pink, at times blotchy and purple; on some occasions there had been a pink rash over the body. The child had been irritable and sleepless and had been apt to sweat freely. The bowels were constipated.

Examination at the time of admission revealed that the child was in poor general condition and was dehydrated. The tonsils were large and injected. Photophobia and muscular hypotonicity were pronounced. The toes and fingers were pink.

After admission the pink colour gradually faded and the muscle tone improved. Photophobia varied in intensity day by day, but gradually became less. The child suffered from bronchitis, which was more severe while teeth were erupting. The weight was increasing and the progress was considered satisfactory. At the time of the meeting the child was receiving a full mixed diet, "Radiostoleum" in a dose of one cubic centimetre twice a day, and orange juice in a dose of 30 cubic centimetres (one fluid ounce) a day. He was also receiving sun baths. A 10% solution of "Argyrol" was instilled into each eye every four hours.

Dr. Robertson's next patient was a male child, aged eight and a half months, who had been admitted on June 10, 1934. He was an only child, and his condition at birth had been poor, his birth weight having been 2·25 kilograms (five pounds). His mother had suffered from *ante partum* hæmorrhage. He had suffered from a disorder described as "indigestion" since birth. His diet had consisted of "Lactone" syrup milk, "Vi-Lactogen", and, at the time of admission, cow's milk. His illness commenced one month prior to admission, with constipation, vomiting, coughing and restlessness. The child was backward and unable to sit up. Examination revealed a thin hypotonic child in poor general condition with subnormal mental development. He was very miserable. The rib junctions were beaded. There were a few râles at the base of the left lung.

Photophobia became pronounced after the patient's admission. The hands and feet were cold and pink at times. Bronchitis recurred with teething. The weight fluctuated for a time, then gradually increased; it was increasing at the time of the meeting. The muscle tone and the general condition had improved greatly. The treatment was the same as for the previous patient, save that adrenaline solution was given in a dose of 0·12 cubic centimetre (two minims) every four hours.

Dr. Robertson said that he was very glad to be able to show the patients with pink disease, because they illustrated very well indeed what had come to be recognized as the important features of the illness. The symptom which seemed to be common to them all was the temperamental change shown by the utter misery. They illustrated also that all cases of the disease were not of the same type. Some came to full development much more quickly than others, ran a much more rapid course, and arrived at the stage of improvement within a few weeks. Other cases were slow to develop, ran a prolonged chronic course and took months sometimes before definite improvement occurred. All the patients shown illustrated a very common observation, that although the appetite might be poor and the child therefore difficult to feed, the food taken was almost always perfectly digested, and very seldom were there any digestive upsets. Photophobia was well seen in one case; this symptom might show itself and disappear several times in the course of the illness. No success had attended attempts to cultivate any micro-organism from the conjunctiva. The actual cause of the disease was not known; but something was known about it. The disease developed in children fed on apparently every possible diet and when there was no deficiency of any known vitamin. The copious sweats, the red, but cold, extremities, the rashes, the loss of tissue turgor, the shedding of the hair and sometimes the teeth, and the absence of reflexes in many cases pointed to nervous disturbances. The finding by Greenfield of a round-celled infiltration of the peripheral nerves, the fact that pyrexia was common, and a second attack of the disease (as far as Dr. Robertson knew) unknown, pointed to an infection, probably by a virus. Dr. Robertson said that his feeling was that pink disease would eventually be found to be one of the epidemic nervous diseases like poliomyelitis, encephalitis *et cetera*, and to have the same method of spread—through a long series of carriers, who were not susceptible, to a particularly susceptible person, who acquired the disease. Dr. Robertson said that the line of attack he should like to see investigated was by immune serum. He thought it would be well worth while, because some children died of the illness, those who recovered suffered weeks of misery, and some were left crippled by bronchiectasis, which occurred in a proportion of the cases. At present nothing much was being done and on the evidence it would appear logical to try the effect of immune serum.

Congenital Malformation of the Heart.

Dr. Robertson also showed a male infant, aged four months, who had been admitted to hospital on October 19, 1934. He was the surviving member of prematurely born twins. His weight at birth was 1·1 kilograms (two pounds nine ounces). For one month prior to admission he had been subject to convulsive paroxysms, in which he gasped for breath, became cyanosed, clenched his hands and rolled his eyes. The paroxysm lasted for about two minutes; after it the child appeared to be exhausted. The frequency of the paroxysms had increased until there was one every hour.

At the time of admission the child was in good general condition; there was no cyanosis; there was a loud murmur

to be heard over the precordium, replacing both heart sounds; there was exophthalmos; the pupils reacted to light; there were nystagmus and strabismus. By X ray examination it was seen that the heart shadow was within normal limits, and there was no apparent abnormality in the skull. Examination of the epiphyses and costo-chondral junctions revealed moderately advanced rickets with a healing reaction. The urine was normal. The red blood cells numbered 4,000,000 and the white cells 10,000 per cubic millimetre; the hæmoglobin value was 60% and the colour index 0·75; the neutrophile leucocytes were in the proportion of 60% and the lymphocytes 38%. The optic disks were pale and the patient did not seem to be able to see.

During the child's stay in hospital the epileptiform seizures gradually became less severe and eventually ceased as a result of the administration of sodium bromide. The weight increased by 0·9 kilogram (two pounds) in eight weeks.

DR. C. A. MCHARDY showed a male infant, aged four months, suffering from a congenital cardiac abnormality. He had not been blue when born. He had suffered from vomiting when first brought home after his mother's confinement. There were three other children in the family; all were well. There was a history that the patient sighed frequently, but was gaining weight and did not cry much or become blue when he cried. A loud murmur was audible over the precordium. A provisional diagnosis of patent *ductus arteriosus* had been made.

DR. W. C. PETHERBRIDGE showed a girl, aged nine months, who had suffered from bronchitis as a sequela of pertussis. The mother had suffered from sepsis in the puerperium and the child had remained in the Women's Hospital from the third day to the sixth week and had not thriven. Examination revealed bronchitis, pallor and great weakness; the extremities were cold and slightly blue; the child was dyspnœic and became distressed by handling; there was a systolic murmur over the precordium, most evident over the valvular areas and to the left of the sternum. During her stay in hospital the child suffered from slight diarrhœa and vomiting at times. She was responding to digitalis. Dr. Petherbridge remarked that the case illustrated that weak hearts produced pallor as well as cyanosis, also that signs on auscultation were easily missed in a restless child.

Achondroplasia.

DR. D. G. R. VICKERY showed a patient suffering from achondroplasia and discussed the features of this disorder. He pointed out that the proximal bones of the limbs were proportionately shorter than the distal; the hands often did not reach to the great trochanters when extended at the sides. The shafts of the bones were firm, there was excess of periosteal bone, and there were prominent ridges for the attachments of muscles. The diaphyses of the long bones were expanded at their ends and fitted cup-like over the heads of the epiphyses. The skull was enlarged and brachycephalic, suggesting hydrocephalus; the frontal region was bulbous, and the fontanelles were patent; the root of the nose was depressed; the chin and lower jaw were well developed. The hands were broad and very short; the fingers were thick and conical and set like the spokes of a wheel ("trident hand"). There were furrows round the limbs, suggesting that the soft parts were too large for the bones. The buttocks were prominent; there was lordosis; and the gait was wabbling. The mental development was equal to the average for the age.

Amyotonia Congenita.

Dr. Vickery's next patient was a female child, whom he had first seen on November 22, 1933, at the age of ten months. The weight at birth had been 3·15 kilograms (seven pounds); the weight at the time of examination was 10·35 kilograms (23 pounds). The child was breast fed. The mother stated that the child was unable to sit up and that from the fourth to the ninth month she had had frequent nocturnal paroxysms in which she rolled her eyes up and turned blue; these paroxysms were relieved when the child was roused and turned over. There had been no paroxysms during the tenth month. She slept more than normal.

Examination revealed that the baby was exceptionally fat and flabby and did not kick as a normal baby should; she was able to move the distal joints of the limbs, but not the proximal. The limbs could be put into abnormal positions without discomfort; the whole musculature was toneless, and all the joints were exceptionally loose. The skin was moist and had a good texture. The features were not coarse. The tonsils were enlarged. The pulse rate was 120 per minute. The condition was considered to be *amyotonia congenita* or *status lymphaticus*. X ray examination revealed no pituitary abnormality or enlargement of the thymus gland. X ray examination of the left hip joint revealed partial dislocation, with shortening of 1·25 centimetres (half an inch) of the left lower extremity. Thyreoid extract was given in a dose of 0·015 gramme (one-quarter of a grain) three times a day. In January, 1934, the patient looked well; she had lost 0·9 kilogram (two pounds) weight; her pulse rate was 140 per minute; she was able to say individual words, and her mental development appeared to be normal; she had six teeth; hypotonia was still pronounced, and the knee jerks were difficult to elicit. In February, 1934, she was able to sit up. In March she was talking a good deal and was much brighter; the knee jerks were present, but were sluggish. At this stage the thyreoid therapy was suspended. In September, 1934, she was able to feed herself, but she made no attempt to crawl or pull herself up. Hypotonia was still pronounced. The final diagnosis was *amyotonia congenita*.

Cretinism.

Dr. Vickery also showed a girl, aged three years, a cretin, who had been under treatment since the age of five months. The mother stated that the child had enlarging swellings above each collar bone, was not taking notice, and was extremely constipated. Dr. Vickery said that this child had the classical symptoms of cretinism. She had been given small increasing doses of thyreoid extract regularly, save for one period of ten weeks during which the mother had ceased treatment on the advice of a medical practitioner, and during which all the symptoms had returned in an acute form. At the time of the meeting she was taking 0·21 gramme (three and a half grains) of thyreoid extract a day; but she was still somewhat coarse featured, dull and not perfectly normal for her age.

Oxycephaly.

Dr. Vickery's last patient was a male infant, aged eight months, who had been examined first on September 11, 1934. The mother had noticed a prominence of the skull in both temporal regions ever since the birth; the prominence was most pronounced on the right side. The child had been born three weeks before full term and the weight at birth had been 3·37 kilograms (seven and a half pounds). The weight at the time of examination was 8·7 kilograms (nineteen pounds six ounces). The development appeared to be normal. The child had sat up at the age of seven months; he took notice of everything and appeared to have a normal mental development for his age. He was a healthy, bright baby, with pronounced flattening of the face. The eyes were set wide apart and there was a broadening of the intertemporal region, there being a more pronounced bulging on the right side. The head was short antero-posteriorly, with flat, towering forehead and occiput; the fontanelles were all closed. The mouth was large, with broad alveolar margins and a narrow, high palate. There were no deformities of the hands or feet. One other child of the family had slight broadening of the intertemporal region; it had been born with torticollis (now cured). The mother was said to have a very small pelvis. Dr. Vickery made a provisional diagnosis of oxycephaly.

DR. J. E. SHERWOOD showed a male child, aged three months, who had been admitted at the age of four weeks,

on September 1, 1934. He was the fourth child of the family; labour had been prolonged and difficult, forceps delivery having been necessary. The weight at birth was 4·5 kilograms (ten pounds), and at the time of admission 3·6 kilograms (eight pounds). The child was breast fed. The second child had had jaundice twenty-four hours after birth, but now, at the age of four years, was healthy; there was no history of undue bleeding in the family.

The history was that twenty-four hours after birth the patient had become jaundiced and had remained so; but the degree of jaundice had diminished somewhat. During the nine days preceding admission to hospital subcutaneous hæmorrhages had occurred; the nose had bled occasionally. Vomiting had occurred at times. The mother had noticed that the neck and limbs were stiff.

The child was very thin and there was some degree of dehydration. There was an icteric tinge of the skin, mucous membranes and conjunctivæ. There were subcutaneous hæmorrhages of the face, trunk and limbs. The neck was rigid. The limbs were spastic.

Intramuscular injection of the mother's whole blood was given and no further hæmorrhage occurred. The jaundice gradually faded. The spasticity of the limbs and neck became more pronounced for a short time, but decreased later. The child cried a great deal. Frequent pyrexial attacks with increase in the pulse rate occurred. While breast feeding was continued the child gradually lost weight, and his stools were frequent, green, relaxed, and sometimes fluid; but the weight increased and the stools became normal when artificial feeding was instituted. There was concomitant pyelitis.

The mother's blood serum did not react to the Wassermann test. On November 3, 1934, the child's blood coagulation time was 25 minutes; the red blood cells numbered 3,000,000 per cubic millimetre, the hæmoglobin value being 50% and the colour index 0·8; the leucocytes numbered 12,000 per cubic millimetre, 68% being polymorphonuclear cells and 32% lymphocytes; there was no diminution in blood platelets; the red cells were deficient in hæmoglobin content and were irregular in size and shape. On November 15, 1934, the red cells numbered 4,500,000 and the white cells 14,000 per cubic millimetre; the hæmoglobin value was 80% and the colour index 0·9; the proportion of polymorphonuclear cells had now decreased to 56%, and there were 2% of immature cells; the red cells were normal in size and shape, and no nucleated red cells were seen. On the same date 15 cubic centimetres of clear fluid under greatly increased pressure were obtained by lumbar puncture; there was no increase in the cell content; no organisms were seen.

X ray examination of the skull and spine revealed no abnormality, nor was evidence of bronchitis found in the lungs.

In addition to the injection of mother's blood, 1·3 cubic centimetres (20 minims) of "Hepasol" three times a day, 0·2 cubic centimetre (three minims) of "Radiostoleum" twice a day, 0·2 cubic centimetre (three minims) of "Adexolin" twice a day, and a citrate of potassium mixture, followed by hexamine, were given. Breast feeding was allowed for eight weeks, then condensed milk was given; at the time of the meeting the strength of the condensed milk solution was 1 in 8, 200 cubic centimetres (six and a half fluid ounces) being given every four hours.

Cerebellar Ataxia.

DR. T. W. BURGESS showed a female child, aged seventeen months. The patient was the second child of the family. The other child had rickets. Birth and development had been normal. The mother had noticed that since the child had begun to walk (two and a half months before) she had been falling down when excited, that she threw her left foot inwards and had a staggering gait. She had been vomiting for two days and had vomited occasionally since birth.

The child's general condition was good. The left leg tended to be thrown inwards. The gait was unsteady. Dr. Burgess suggested that the patient was suffering from cerebellar ataxia.

(To be continued.)

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Stuckey, Edward Seavington, M.B., B.S., 1933 (Univ. Sydney), 57, Spit Road, Mosman.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Boan, Stanley, M.B., B.S., 1934 (Univ. Melbourne), Alfred Hospital, Prahran, S.1.

Clarke, Edmond, M.B., B.S., 1932 (Univ. Melbourne), Mooroopna Hospital, Mooroopna.

Donald, Charles Douglas, M.B., B.S., 1933 (Univ. Melbourne), 448, Glen Eira Road, Caulfield, S.E.8.

Gepp, Dorothy Marian, M.B., B.S., 1933 (Univ. Melbourne), Children's Hospital, Carlton, N.3.

Heseltine, Mary Jermyn, M.B., B.S., 1934 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Hutchings, Mildred Margaret, M.B., B.S., 1934 (Univ. Sydney), Melbourne Hospital, Melbourne, C.1.

Kidd, David Alan, M.B., B.S., 1933 (Univ. Melbourne), Beechworth Mental Hospital, Beechworth.

Pern, Geoffrey, M.B., B.S., 1933 (Univ. Melbourne), Prince Henry's Hospital, Melbourne.

Provis, Ethel Lillian, M.B., B.S., 1932 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Rank, Benjamin Keith, M.B., B.S., 1934 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Van Nooten, George Henry, M.B., B.S., 1929 (Univ. Melbourne), Alfred Hospital, Prahran, S.1.

Correspondence.

THE TRAINING OF SURGEONS.

SIR: Though I have decided views on the recent medico-ministerial controversy, it is not my present intention to take part in it, but to suggest a practical solution of the only important point at issue, that is, the alleged incompetence of newly qualified surgeons.

I suggest that junior resident medical officers at the large hospitals, under supervision, be allowed to commence and, at the discretion of the supervisor, to complete, 40% of certain specified major operations undertaken in the public portions of those hospitals; that a further 10% or more be performed by senior resident medical officers; and that knowledge of this arrangement be made public.

Such a scheme could be amplified. The surgical degree could be withheld until a certain number of operations had been performed. This would leave the newly qualified man free to practise immediately, as a physician only, if necessary.

The advantages are many and varied:

1. Every future surgeon must do his first operation somewhere. Is it not desirable that he should do it under supervision?

2. Every country practitioner should feel capable of opening an abdomen, and it is unfair that the necessary experience should be obtainable only by patience and ingenuity and by grovelling to hospital boards and honoraries, as at present.

The last small, but important, part of a man's surgical qualification should not be left for him to obtain by his wits.

The fact that a man has received a surgical degree without ever having made an incision in his life, and even more so the fact that, in spite of a residency, he has had no opportunity of doing so, is a disgrace to an otherwise superlative system of surgical education and is due in some part—with one or two notable exceptions—

to the selfishness of honorary surgeons. They must face the fact that the country must and will have surgeons, even if they have to train themselves.

3. It would do a great deal toward solving the financial difficulties of the hospitals, as applications for intermediate surgical beds would rise rapidly.

4. Prominent surgeons would be paid for a greater proportion of their work.

5. Each hospital would soon develop one or two highly skilled surgeons among their senior resident medical officers, who, as operators and supervisors, would be laying a very sound foundation for their surgical careers.

Much "assisting" at operations is useful clinical experience in one way, but not until the assistant sets out to perform the operation himself does he realize the depth of his own inability.

Yours, etc.,

G. B. DOWNES, M.B., Ch.M.

"Ardlui",
Parkes,
New South Wales,
February 9, 1935.

TREATMENT OF SEPTIC STATES ASSOCIATED WITH PREGNANCY.

SIR: Some points in Dr. Worrall's criticism of my paper (THE MEDICAL JOURNAL OF AUSTRALIA, February 9) seem to call for a reply.

1. If there was one thing more than another which I hoped I was making clear, it was that I considered it bad practice to attempt to empty a uterus of the products of conception through an undilated cervix; in fact, it was, I thought, just in this condition that I strongly advocated: (*a*) If bleeding were present, vaginal packing to stimulate the uterus to empty itself. I do not think that this checks drainage from the uterus owing to its stimulation of the contractile muscle action. Does one not frequently find the whole ovum lying free in the top of the vagina twelve hours later? It may prevent drainage from the vagina which does not matter, for Fairbairn (I think) has proved that absorption from the vaginal mucous membrane is practically negligible. (*b*) If bleeding is not alarming, pituitrin or quinine, preferably the latter. I am sorry if this position was not clearly stated.

2. I do not doubt that, in the hands of so experienced and expert a surgeon as Dr. Worrall, the sharp curette is perfectly safe. I should use it myself more often if it were not for example in teaching. Where I feel that it may be dangerous is in the hands of men who obviously have not the same constant opportunities of dealing with these cases as the public hospital surgeon, and who may be tempted to use it too easily in just those cases in which Dr. Worrall himself admits interference is dangerous.

3. My advocation of waiting "for obvious signs of pus formation" in parametric inflammation was due to the fact that, in my experience, a fairly large proportion of these cases do clear up even without drainage—surely a desirable happening. And the interesting fact is that the more cases I leave thus, the greater the proportion becomes.

4. My reason for getting further and further away from Dr. Worrall's teaching (he is quite correct in assuming it was his teaching to which I referred) is that I think again that quite a large proportion of these cases, after drainage, return to normal health without abdominal section, and I, in my present state of ignorance, cannot tell which of them will do so and which will not, except by "trial and error". I cannot agree with Dr. Worrall's words that the focus will "in all probability" flare up again. Some obviously will, and these are operated on; others (and this is the point), and I think a surprising number, will not, and I prefer to give them this chance, for, if operated on, they stand a good chance of being sterilized, if not unsexed.

5. I have never seen a case of stenosis follow cauterization of the cervix by the electric cautery, which is the form I advocate rather than diathermy.

Finally, sir, I am very grateful to Dr. Worrall for his kindly criticism and his still more kind appreciation. I have been and am greatly indebted to him for many useful lessons learnt from watching his work and from reading his writings, and it is with trepidation that I dare differ from him. But, although my paper was expressly written to be read in a country centre, where the hearers would mainly be men without the opportunity of much hospital experience in this type of work and consequently was planned on conservative lines, I did not withdraw any of that conservatism in preparing it for publication, for I felt that conservatism is the right note for the time.

Yours, etc.,

RUPERT E. MAGAREY.

North Terrace,
Adelaide,
South Australia.
February 18, 1935.

THE WHITE MAN IN THE TROPICS.

SIR: Sir James Barrett's letter in your issue of February 16 last is phrased in a way that is inadvertently liable to misinterpretation. Obviously, in saying that "from the Queensland border to the Indian Ocean north of the Tropic of Capricorn there are 6,400 white people in this enormous territory" *et cetera*, Sir James refers to the western boundary of Queensland and excludes that State. Indeed, the later context makes this obvious, but it has been interpreted by a correspondent of mine, and therefore doubtless by others, as meaning that Queensland is included. Actually it would have been necessary to have added to the 6,400 quoted the 240,829 (1933) people who reside in the tropical portion of this State, a population approximating that of the whole State of Tasmania.

As long ago as 1921 the population on and above the Rockhampton-Longreach line was 181,397, so that the rate of increase has been about 2·7% yearly during those twelve years, as against a rate of 1·5% for all Australia. The whole population of Queensland in 1933 (and it must be remembered that all Queensland lies above 28° S.) was 945,706. Practically a million people, therefore, live here in latitudes and areas that have during a century demonstrated by their actual results the futility of the theoretical and philosophic approach to the practical problem of tropical colonization. (In 1852 the importation of Eurasions to work round Brisbane in the most south-westerly corner of the State was flatly negatived on the historical and philosophical grounds that, having some white blood, they could not possibly bear the strain of labour there!)

Dr. Grenfell Price's excellent address, published in THE MEDICAL JOURNAL OF AUSTRALIA of January 26, is welcome as being entirely in line with the views held in Queensland and as indicating his frank avowal of the advance he has made in that direction since his delivery of the Macrossan Lectures in Brisbane some years ago.

Dr. Hone, in his comment, however (page 129), draws a distressing picture of the medical philosopher who is impelled from a range of 2,000 miles to decide as to the proper *minutiæ* of life and circumstance for the tropical resident of Darwin. Fortunately we in Queensland do not need to rise heavily from the table equally depressed after a reading of Ellsworth Huntington's "pessimistic" writings or my own "optimistic" papers. We have the advantage of the actual experience of many years. I can assure Dr. Hone that during the lifetime he has spent in South Australia many hundreds of thousands of white men have put the matter to a surer test, with results that demonstrate my "optimism" to be entirely justified. Almost a quarter of a million of them have been successful in selecting by actual test the areas suitable to settlement, and the process is going on at a more rapid rate than anywhere else in Australia. As Dr. Grenfell Price pointed

out in his Macrossan Lectures, the Northern Territory and probably north-west Western Australia would have progressed at an equal rate in the areas suitable to settlement had it not been for South Australia's successful obstruction to the only possible means of expansion—latitudinal rather than longitudinal. Dr. Hone's suggestion that the medical profession might stimulate activity along a scientific line anticipated and perhaps was inspired by the new Commonwealth activity that has been obvious during the last couple of months. Had it come twenty-five years earlier, following Breinl's original work (the Commonwealth took over in 1911), or even ten years ago, following the publication of my own "White Man in the Tropics", practical considerations would have long since established the link missing to economic success—proper communications. Aviation has rendered the development of the north inevitable. The next few years will show whether and to what extent the dead hand of the south-east corner of the continent will permit itself to be infused with an economic life-stream flowing in from some other portal than Adelaide, Melbourne or Sydney.

Yours, etc.,

Brisbane, February 20, 1935. RAPHAEL CILENTO.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on February 4, 1935.

The following degrees were conferred *in absentia*:

Bachelor of Medicine (*M.B.*) *and Bachelor of Surgery* (*B.S.*): Herbert James Birchley, Gregory Stephen Flynn, Ronald George Lyne, Alfred John Morton, Ann Fraser Thompson.

Bachelor of Medicine (*M.B.*): Patrick Francis Murphy.

The Senate was advised of a bequest of £208 7s. 10d. to the Cancer Research Fund, and the legacy has been received from the Perpetual Trustee Company, Limited (executor of the will of the late Mrs. Alice Rosanna Pearce).

A gift from the British General Electric Company, Limited, of a showcase for the Veterinary Science School was accepted with grateful thanks.

The following appointments were approved: Mr. R. M. Kirkpatrick, B.D.S., as Lecturer in Dentistry; Mr. H. R. Kemp, B.D.S., as Honorary Tutor in Oral Surgery in the Dental School; Mr. I. de V. W. Montgomery as House Surgeon in the Department of Veterinary Science.

Congresses.

LIGUE INTERNATIONALE CONTRE LE RHUMATISME.

THE *Ligue Internationale contre le Rhumatisme* will hold its fifth international congress at Lund (Sweden) in September, 1936. Professor Dr. S. Ingvar, Professor of Internal Medicine in the University at Lund, has been elected chairman, and Professor Dr. G. Kahlmeter, Stockholm, secretary.

Further information may be had from the Secretary of the Ligue, Dr. J. van Breemen, Keizersgracht 489, Amsterdam.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1925 *to* 1933, of Queensland, as duly qualified medical practitioners:

Agnew, Ainslie Francis, M.B., B.S., 1926 (Univ. Melbourne), Ipswich.
Bryant, Vincent Julian, M.B., Ch.M., 1925 (Univ. Sydney), Coolangatta.
Glastonbury, Kevin, M.B., B.S., 1926 (Univ. Adelaide), Brisbane.
Hill, Lettie Grace, M.B., B.S., 1930 (Univ. Melbourne), Brisbane.
Lally, Muire Geraldine, M.B., B.S., 1934 (Univ. Melbourne), Brisbane.
Mitchell, Paul Wanostrocht, M.B., Ch.M., 1914 (Univ. Sydney), D.P.H., 1918 (Univ. Sydney), Brisbane.
Rickard, Haymond Victor, M.B., 1924 (Univ. Sydney), F.R.C.S., 1930 (England), Kingaroy.
Row, Edward Peter, M.B., B.S., 1933 (Univ. Sydney), Brisbane.
Sheil, Ernest Richard Glenister, M.B., B.S., 1931 (Univ. Sydney), Innisfail.
Theile, Egmont Friedrich Hellmuth, M.B., 1933 (Univ. Sydney), Brisbane.
Whiddon, Helen Maude, M.B., B.S., 1933 (Univ. Sydney), Brisbane.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the Medical Act, 1918, of Tasmania, as duly qualified medical practitioners:

Stewart, Dorothy Marion, M.B., B.S., 1934 (New Zealand), General Hospital, Hobart.
Carter, James Leontine Roy, M.B., B.S., 1925 (Univ. Melbourne), Public Hospital, Launceston.

MEMORIAL TO THE LATE NEVILLE HOWSE.

THE Victorian medical officers, both of the Army Medical Corps and those who belonged to the Australian Imperial Force, have proposed the purchase of a bronze casting of a bas-relief bust of the late Neville Howse, modelled by Miss Rose M. Phillips, of Vaucluse, Sydney. Permission has been given for this to be placed in the museum of the Australian Institute of Anatomy at Canberra, in one of the spaces reserved for distinguished medical men. If any other friends of Neville Howse desire to participate in this memorial, they are asked to forward their contributions to Eric Gutteridge, 12, Collins Street, Melbourne, C.1. It is suggested that the maximum subscription be ten shillings and sixpence.

Books Received.

EXPERIMENTAL BACTERIOLOGY IN ITS APPLICATIONS TO THE DIAGNOSIS, EPIDEMIOLOGY AND IMMUNOLOGY OF INFECTIOUS DISEASES, by W. Kolle and H. Hetsch, translated from the seventh, completely revised, German edition by D. Erikson, the English version, incorporating further revision, edited by J. Eyre, F.R.S.E., F.Z.S., M.D., M.S., D.P.H.; Volume II; 1934. London: George Allen and Unwin. Super royal 8vo., pp. 613, with 118 plates and 200 text figures. Price: 30s. net.

Diary for the Month.

MAR. 19.—Tasmanian Branch, B.M.A.: Council.
MAR. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAR. 20.—Western Australian Branch, B.M.A.: Branch.
MAR. 22.—Queensland Branch, B.M.A.: Council.
MAR. 26.—New South Wales Branch, B.M.A.: Council, Quarterly.
MAR. 27.—Victorian Branch, B.M.A.: Council.
MAR. 28.—South Australian Branch, B.M.A.: Branch.
MAR. 28.—New South Wales Branch, B.M.A.: Annual.

Medical Appointments.

Dr. A. M. Cudmore (B.M.A.) and Dr. P. S. Messent (B.M.A.) have been appointed Members of the Dental Board of South Australia, under the provisions of the *Dentists Act*, 1931.

* * *

Dr. A. A. Merritt (B.M.A.) has been appointed Medical Officer of Health by the Murray District Road Board, Western Australia.

* * *

Dr. G. A. Murray has been appointed a Commissioner for Declarations, Perth, Western Australia, under the *Statutory Declarations Act*, 1911-1922.

* * *

Dr. R. J. O'Connor (B.M.A.) has been appointed Pathologist and Micro-Biologist, Adelaide Hospital, South Australia.

* * *

Dr. C. R. O'Brien (B.M.A.) has been appointed Junior Medical Officer, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. G. W. Pottinger has been appointed District Medical Officer at Roebourne, Western Australia.

* * *

Dr. K. S. M. Brown (B.M.A.) has been appointed a Member of the Boards of Official Visitors to the Mental Hospitals at Parramatta and Rydalmere, New South Wales.

* * *

Dr. H. V. Hanson has been appointed Medical Officer, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. H. G. Anderson (B.M.A.) has been appointed Certifying Medical Practitioner at Meeniyan, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. H. J. Enniss (B.M.A.) has been appointed Certifying Medical Practitioner at Wonthaggi, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiv to xvii.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Medical Officer.
BLAIR ATHOL DISTRICT HOSPITAL, BLAIR ATHOL, QUEENSLAND: Medical Officer.
CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officer.
MENTAL HOSPITAL, NEW NORFOLK, TASMANIA: Assistant Medical Officer.
PUBLIC SERVICE BOARD, SYDNEY, NEW SOUTH WALES: Medical Officer.
REPATRIATION GENERAL HOSPITAL, CAULFIELD, VICTORIA: Visiting Physician.
THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 267, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.

XUM